AURICULAR FIBRILLATION AND ITS RELATIONSHIP TO CLINICAL IRREGULARITY OF THE HEART.

By THOMAS LEWIS.

Reprinted from "Heart."
Vol. I, No. 4, March, 1910.
Pages 306 to 372.

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It will be shown that the type of irregularity, which is one of the chief features of the condition, is the commonest persistent irregularity exhibited by the human heart, constituting as it does approximately 50 per cent. of all such cases; and it will be demonstrated that the disturbance of cardiac rhythm is to be sought in the auricle and attributed to temporary or permanent inco-ordination of the musculature of that chamber.

In brief, evidence is forthcoming that of the numerous examples of persistent irregularity of the heart familiar to clinicians, approximately 50 per cent. are the result of auricular delirium or fibrillation.*

Our acquaintance with the facts upon which this final conclusion rests is the outcome of the studies of a large body of workers. A retrospective survey of the observations upon clinical material permits the recognition of cases, belonging to the category discussed, over a period of many years. Fully possessed of the facts, we may trace the earlier descriptions of the condition along two lines ; and it is mainly upon these separate paths that the course of observation has been pursued, and pursued until quite recent years by distinct investigators. The earliest graphic observations were carried out independently upon the arterial and venous systems. The two paths have converged and finally have met during the last decade. For this union of the facts derived from two separate sources and for the wide range of supplementary knowledge relating to the condition, we are indebted almost exclusively to the introduction of the new graphic methods of examining the cardiac mechanism which are now at our disposal.

On the one hand a markedly irregular pulse, especially associated with mitral disease in its later stages, was the subject of study by mechanical means from the epoch marked by the introduction of the sphygmograph. It is portrayed by Marey[11], Riegel[53b], Sommerbrodt[50] and many other writers. It has been termed the "mitral pulse," and has been attributed amongst other causes to "delirium of the heart." It has passed by the name of *Pulsus arhythmicus* (Janowski[25]), and by the name *Pulsus irregularis* (Riegel[53b]). It has been identified, in a classic but obsolete nomenclature, with the adjectives *irregularis*, *inæqualis*, *deficiens* and *intermittens*.

On the other hand, a prominent systolic pulsation in the veins of the neck was described by Bamberger[1], Skoda[59] and others, and was attributed, and has long since been held as due, to tricuspid incompetence. The subjective timing of the venous pulsation was endorsed by Riegel[53a], who obtained the first graphic records of the movement; but the class of case in which such pulsation is found essentially was not isolated, neither was its full significance grasped, until the more exact and more applicable technique of Mackenzie was introduced.

It is since the year 1902, when the "Study of the Pulse" was published, that chief progress has been made. It is to Mackenzie that we owe the definite co-relation of the two phenomena, gross irregularity of the heart and the systolic venous movement, which he has termed the "ventricular form of venous pulse." In the work referred to[31], this writer first demonstrated their frequent association and ascribed them both to a single underlying condition, namely, paralysis of the auricle. A year later Hering[15], describing

* Preliminary notices of the observations have appeared[31 & 32], and examples of the curves were demonstrated before the Medical Society of London on 13th December, 1909, and before the Pathological Section of the Royal Society of Medicine on 4th January, 1910, at University College Medical School.

the arterial pulse alone, laid more stress upon its characteristics and spoke of it under the title *pulsus irregularis perpetuus**. He appears to claim[17] to have implied, by the use of this term, a specific type of rhythm, but the facts brought forward in his paper failed to carry conviction of an irregularity *sui generis*. The recognition of its specificity has been arrived at gradually and the facts supporting the conclusion have been contributed largely by Mackenzie. In particular, the emphasis which he laid upon its frequent association with the ventricular form of venous pulse in 1904[35], and the prominence given to this observation in a later paper, based upon an examination of 500 cases[37], must be noticed. But in reality it is only since the galvanometric examination of the heart has been available that the probability of its specific nature has grown to certainty.

In his papers of 1904-5, Mackenzie[35 & 36] brought forward several new and important facts, and most striking amongst them, in the light of our present knowledge, were evidences that the auricle is active. Formerly regarding the auricle as paralysed, because no sign of activity could be found, he attempted at this time to separate a special group of cases in which auricular activity was considered probable. Auricular activity was assumed, (1) because the auricle was found hypertrophied at autopsy; and (2) because certain instances were observed in which the normal rhythm reasserted itself. It is to these papers more especially that we are indebted for the observation that in all cases of complete irregularity of the heart there is an entire failure of signs of the *normal* auricular contraction during diastole; and further, for the first record of cases of this nature, in which it is probable that little dilatation of the right heart and little triscuspid regurgitation is present. His earlier view that the condition results from auricular distension as a consequence of valve incompetence was at least partially abandoned, and the rhythm was ascribed as the cause rather than the result of the eventual dilatation. In 1904 Mackenzie postulated the view which he has since held, that in many cases the seat of the rhythm is in the junctional fibres lying between auricle and ventricle, and by conceiving the simultaneous contraction of auricle and ventricle in response to impulses from this single source attempted to explain the absence of every sign of normal auricular contraction which he had demonstrated to be one of the chief features of such cases. In 1906 Hering[16] realised the correctness of Mackenzie's assertion that the completely irregular pulse when once established is not of necessity perpetual. In 1907 Theopold[62] described cases confirming the view that the rhythm is not a secondary phenomenon to tricuspid leakage.

In 1907-8 Mackenzie[37 & 38] adopted the hypothesis of the nodal origin of the rhythm more generally, holding the node of Tawara† to be the seat of

* The qualification "perpetual" has since been discarded.

† A specially differentiated structure at the auricular side of the junctional tissues between auricle and ventricle.

disturbance in all cases of complete irregularity found in combination with the systolic form of venous pulse. He therefore included all such cases under the term "nodal rhythm."*

Hering[18] has recently added evidence of the supraventricular formation of the ventricular impulses.

Such is the history of the main steps in the attempt to unravel the problem of the mechanism upon which the symptomatology is dependent, and, for the time being, this brief account will suffice. Papers relating to the subject have been prolific, a mass of detailed knowledge has accumulated, discussion has been unrestrained and speculation has oftentimes exceeded the limit warranted by the facts. Further reference to the work of previous observers will be made more conveniently under the various headings and sub-headings which follow.

A general account of complete irregularity of the heart.

The following account of the condition is based upon a close study of seventy-three cases from all of which simultaneous radial and venous curves, and from thirty of which electrocardiographic curves, have been taken. The clinical polygraph and Mackenzie's ink polygraph, fitted with specially light writing points and arranged with a considerable reduction in the air content of the transmission apparatus, have been employed in obtaining records of pulsating areas. The electrocardiograms have been obtained with Edelmann's large pattern of the string galvanometer of Einthoven. During the earlier days when the work was in progress, the electrodes consisted of baths of salt solution in which zinc terminals were immersed, but the greater part of the investigation has been carried out with non-polarisable electrodes consisting of zinc rods immersed in saturated zinc sulphate solution, the last held in a porous jar itself lying in the salt solution. Simultaneous radial or venous and electrocardiographic curves have been secured by adding parts of the Mackenzie polygraph to the galvanometric outfit. The patients were always in the same room with the apparatus while the records were taken.

Frequency.

Complete irregularity of the heart is a very common condition. During two years of observation 114† cases of persistent‡ irregularity of the heart

* On the Continent the rhythm is variously described as "chronic arhythmia," "arhythmia perpetua," etc.

† I have excluded entirely all cases of irregularity which I have seen since the first announcement of my view of the mechanism present in complete irregularity of the heart. For since that date I am unable to affirm that the cases presenting themselves have been impartially chosen. It is on this account that the number of cases of complete irregularity from which the conclusions as to the mechanism are drawn exceed those mentioned in the statistical account which is now given.

‡ By persistent irregularity I denote cases in which, from time to time, irregularity is a notable feature of the case. Cases in which single extrasystoles at long intervals, or cases in which numerous extrasystoles have been observed only on one or two occasions have been excluded.

have been examined and the mechanism analysed in each case. These cases subdivide themselves as follows :—

1) Complete irregularity of the heart ..	57
2) Ventricular extrasystoles	34
3) Auricular extrasystoles	11
4) Irregularity due to heart-block ..	3
5) Gross sinus irregularities	3
6) Complex irregularities and paroxysmal tachycardias	6

We see, therefore, that of 114 cases 57 were of the form under consideration, constituting 50 per cent. of the total number. With the exception of groups 4 and 6, the cases were in no way selected. All cases of persistent irregularity which have been observed during the course of my out-patient clinic at the City of London Hospital for Diseases of the Chest, and all cases of the same sort to which my attention has been directed amongst the indoor patients at University College Hospital have been included.

Etiology (*based upon* 73 *cases*).

Of the sexes affected, 43 were males and 30 females.

The age limits in this series are 13 to 84. The average age is 41·9 (67 cases).

As regards the remaining etiological features examined, they are included in the accompanying table.

It will be seen that a history of rheumatism* or chorea has been present in 26 cases ; in 3 additional cases at least there was a history of one or other in the family alone. Amongst the remainder mitral stenosis was present in 16, pericardial adhesions in one case, and subacute streptococcic endocarditis in one case. Of 64 cases 29 were of undoubted rheumatic predisposition, or had actually suffered from joint affection or chorea. In addition, in 18 instances the rheumatic taint was open to suspicion. The total of 47 cases may be included in a rheumatic group, and for the most part these patients are amongst the youngest of the series. (The average age of 45 cases of the rheumatic group was 34·2 ; that of 22 cases of the non-rheumatic group 57·7.) 37 cases or 52 per cent. were instances of mitral stenosis, and the relationship to this form of valvulitis is so definite that additional figures relating to it may be given. In dealing specially with mitral stenosis I chose only those cases which came to my out-patient department, for the majority of the cases of mitral stenosis admitted to the

* As a criterion of rheumatism I have taken a past diagnosis of rheumatic fever, or the history of an acute illness with swelling and pain in several joints.

general wards have disordered heart rhythm. Of 72 cases of mitral stenosis collected in this way (in each and all of which curves have been obtained), 57 presented the normal sequence of chamber contraction, and 15 or 20·8 per cent. exhibited complete irregularity of the heart.

DIAGNOSIS.

		Mitral stenosis	Complete irregularity	Arterial disease	Granular kidney	Pericardial adhesions	Aortic disease	Remainder	
Rheumatic or choreic history	Mitral stenosis	21	..	..	..	..	..	..	26
	Complete irregularity	..	3	..	..	..	..	..	
	Pericardial adhesions	..	..	..	..	1	..	..	
	Granular kidney	..	..	..	1	..	..	..	
Rheumatism or chorea in family	Mitral stenosis	1	.	..	..	..	..	..	3
	Complete irregularity	..	2	..	..	..	..	..	
No history of rheumatism or chorea	Mitral stenosis	10	..	..	..	..	..	..	35
	Arterial disease	..	..	6	..	..	..	..	
	Complete irregularity	..	5	..	..	..	..	..	
	Granular kidney	..	..	..	3	..	..	..	
	Aortic disease	..	..	..	..	..	2	..	
	Aneurism	..	..	..	..	..	..	2	
	Emphysema and br.	..	..	..	..	..	..	2	
	Strepto. endocarditis	..	..	..	..	..	..	1	
	Pericardial adhesions	..	..	..	..	1	..	..	
	Tubereulous pleurisy	..	..	..	..	..	..	1	
	Chronic alcoholism	..	..	..	..	.	..	1	
	Pneumonia	..	..	..	..	..	..	1	
Not noted	Mitral stenosis	6	..	..	..	..	..	..	9
	Not noted	..	..	..	..	..	..	3	
		38	10	6	4	2	2	11	73

The symptoms.

For the purpose of studying the symptomatology of complete irregularity of the heart those patients have been selected in whom the irregularity and its concomitant phenomena were alone present. Dilatation and signs of valvular lesion were absent, and the remaining organs appeared to be normal. Cases in which the affection is paroxysmal are also important from this

point of view, and are included. This selection permits the exclusion of certain symptoms which are not referable to the irregularity itself. It must be acknowledged that the subjective manifestations are few in number, and that they may be almost entirely absent. A certain degree of short-windedness during or after exertion, an occasional fluttering in the neck or chest may be experienced. A general feeling of ill-health, often associated with easy exhaustion, is not uncommon. Gastric discomfort and loss of appetite are not infrequent. The symptoms are more prominent in nervous subjects. Such are the main disturbances in cases in which the ailment is of short standing.

There appears to be a special symptomatology, or, speaking more correctly perhaps, a more profound disturbance at the time of the onset of the irregularity. The actual onset has probably never been recorded, but the symptoms and observations at or about the onset all point to its being as sudden as it is in the case of regular paroxysmal tachycardia. In all, one case of onset in which the arrhythmia became persistent and five cases of the paroxysmal type of complete irregularity have come under my own observation. In the first case the irregularity commenced while the patient lay in bed, and shortly after waking. He was seized with violent palpitation in the chest, a choking feeling in the throat, and inability to "catch his breath." The heart was observed by him to be beating very rapidly and irregularly. The symptoms lasted 15 minutes and were relieved by brandy, but the irregularity is said to have continued until he was next seen; it has been present ever since (*CASE 11*). In three paroxysmal cases (*CASES 2* and *10* and Fig. 9) the change from one type of mechanism to the other occurred without appreciable manifestations* of a subjective nature and usually passed unrecognised. In one of these cases there were physical signs pointing to thoracic aneurism and anginal attacks were frequent, but no relationship could be traced between the anginal attacks and the change of heart action. In a fourth paroxysmal case the first attack was accompanied by great prostration, pallor, fainting, and tonic contraction of the arms and legs (simulating tetany). In the fifth case two attacks occurred. The patient was admitted suffering from streptococcic endocarditis, and post-mortem a large ulcerated area was found in the right auricle. The first attack produced great prostration, exhaustion, and violent palpitation. The second attack, which was similar, proved fatal.

The cases which have come under observation show that the paroxysms may last a few hours or several weeks or months. The symptoms, when present, seem to be more marked at the commencement of the irregularity.

The symptomatology in the majority of patients, cases in which obvious valvular disease or dilatation is present, is that of decompensation in its various degrees. It is not in any way identified with the condition of irregularity itself.

* In one of these the change was from an attack of paroxysmal auricular tachycardia to complete irregularity (Fig. 9).

For further accounts of the symptomatology in this condition reference may be made to the writings of Mackenzie, and to a recent paper by Hewlett[22].

The radial pulse curves.

The character of the radial pulse curves in complete irregularity of the heart is so striking that it could not, and as we have seen did not, escape early attention.

Several tracings of the arrhythmia are given by Marey[14] (Figs. 199 to 204) as examples of the pulse in mitral insufficiency. The same type of pulse is figured by Fagge[8] (Fig. 3), Mahomed[13] (Plate IV, Figs. 7, 13 and 16), Walshe[63] (Plate I, Figs. 8, 10 and 11), Sansom[57] (Figs. 112, 122B, 159, 160, 163, etc.), Steell[61] (Figs. 9, 10, 42 to 44, 49, 59, 60, 75, 76, 78 to 82, 86 and 89), and by Broadbent[2] (Figs. 27 and 36). Numerous figures will be found in Mackenzie's books.

The irregularity is of the most varied description. The pulse may be slow or fast, and the variation in rate is great (30 to 200). The beats may be all of small excursion; more commonly there is a haphazard intermingling of forcible and weak contractions, and the latter are often markedly dicrotic. The radial pulse is often but an indifferent index of the rate of the ventricle; many beats are not transmitted. The pulse rate may be considerably reduced, either as a result of "dropped" beats or as a consequence of the actual slow speed of the ventricle. The beats may show coupling over short or long stretches of curve. The fast types are the commonest, and in these the usual rate of the ventricle is approximately double the normal rate (110 to 150). It is usually at these fast rates that the disorderly character of the pulsation is so prominent. With the slower rates the irregularity is not so prominent, nevertheless, it is always present, a fact which can be determined by careful measurement of the tracings. Where the grade of irregularity is high the condition may be recognised by feeling the pulse; and with experience even the lesser grades of disorder, for example those met with in cases where the pulse is slower, can be identified by similar means, though the method is inevitably uncertain. A Dudgeon tracing is adequate in the majority of patients, and the disorder may be recognised by two criteria. First and most important is the absolute character of the arrhythmia. The heart action is never regular, and seldom or never do two beats of the same character or length succeed each other. In a long curve it is rare to find any two short sections of tracing which have even a superficial resemblance to each other. The pauses betwixt the beats bear no relationship to one another, and in this feature the irregularity stands in marked contrast to all other varieties. The second criterion consists in the absence of a definite and continued relationship between the strength of a beat and the length of the pause which precedes it. A strong beat may follow a short pause, and a weak beat may succeed a long pause. A few examples of the

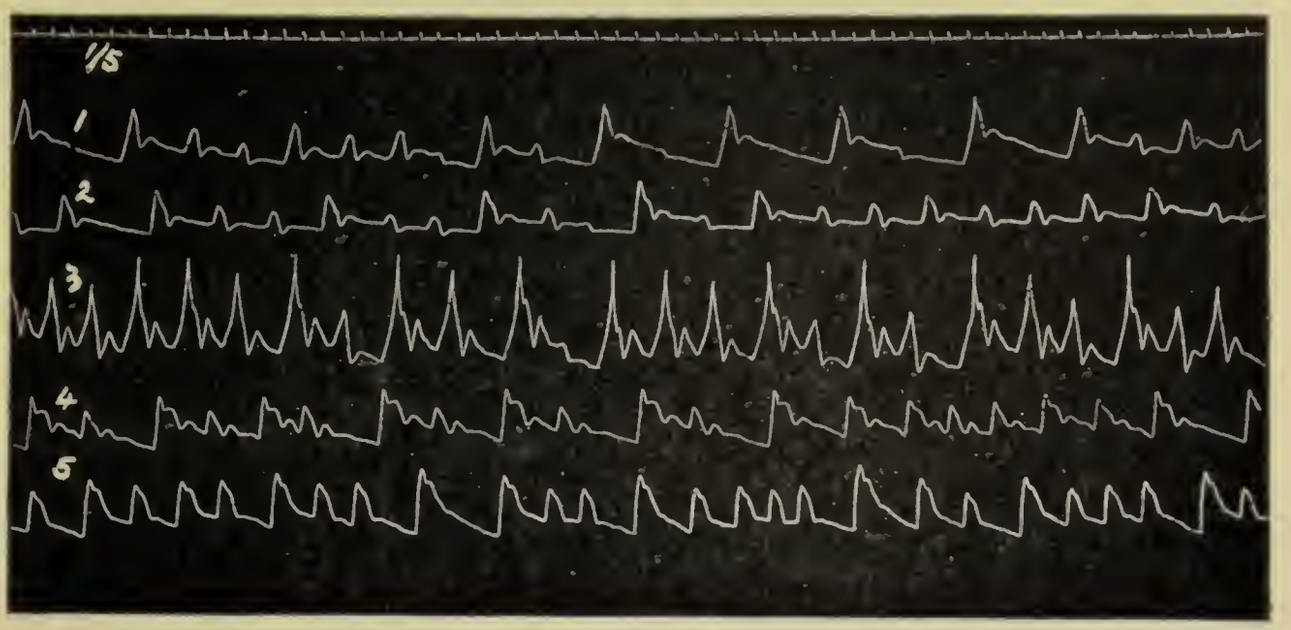


Fig. 1. × $\frac{5}{6}\frac{5}{5}$ linear. Radial pulse curves taken with a Dudgeon sphygmograph. The time tracing, which applies to all curves beneath it, is in $\frac{1}{5}$ sec.. The figure illustrates the general characteristics of the disordered heart action considered in the text.

Curves 1 and 2.

CASE 1.— A. H., a man aged 48, admitted to hospital suffering from mitral stenosis of rheumatic origin and general cardiac dilatation. Enlargement of liver, distended veins and dropsy were present. Irregularity complete and persistent; murmurs early and mid-diastolic; venous curves of ventricular form; electrocardiographic curves of usual type (as in Fig. 14 *I*). The oscillations were maximal when the electrodes were placed over the right auricle.

Curve 3.

CASE 2.— W. S., a man aged 64, the subject of bronchitis, emphysema and arterio-sclerosis. No history of rheumatism. Heart somewhat enlarged to right and left. Heart sounds normal; S. B. P., 150 mm. Hg..* With the exception of shortness of breath on exertion no signs of broken compensation were present. The irregularity disappeared on one occasion for a few days, the pulse regularity was then interrupted by auricular extra-systoles. The *a-c* interval was normal. With the complete irregularity the venous pulse was ventricular in outline, the electrocardiograph was typical, the oscillations were maximal in leads from the parts of the chest wall covering the right auricle. There was no pulse slowing with heavy doses of digitalis.

Curve 4.

CASE 3.—W. P., a man aged 37, suffering from mitral stenosis of rheumatic origin. Heart enlarged to right and left, dyspnœa and slight liver enlargement, no dropsy. Pulse persistently irregular: ventricular form of venous pulse; electrocardiograms typical; oscillations maximal with electrodes in neighbourhood of right auricle.

Curve 5.

CASE 4.—R. N., a man aged 65, suffering from aneurismal dilatation of the whole thoracic aorta, pulmonary œdema, associated with arterial sclerosis, emphysema and signs of sclerotic kidney. Dropsy and liver enlargement present. Pulse persistently irregular; ventricular form of venous pulse. Died unexpectedly.

* This blood pressure reading is a measure of the obliteration pressure of the most forcible beats. Blood pressure estimations in cases of complete irregularity are extremely unsatisfactory; the beats force their way through the armlet at widely varying pressures.

pictures presented by Dudgeon tracings are given in the accompanying figure. They may serve with the brief notes attached to them as a guide to the recognition of the type of case with which we are dealing. They illustrate the main points referred to in the text, but the variety shown is so great that they can scarcely be held even as representative of the irregularities which may occur. Numerous and additional examples are scattered throughout the simultaneous tracings which illustrate this paper.

The venous pulse curves.

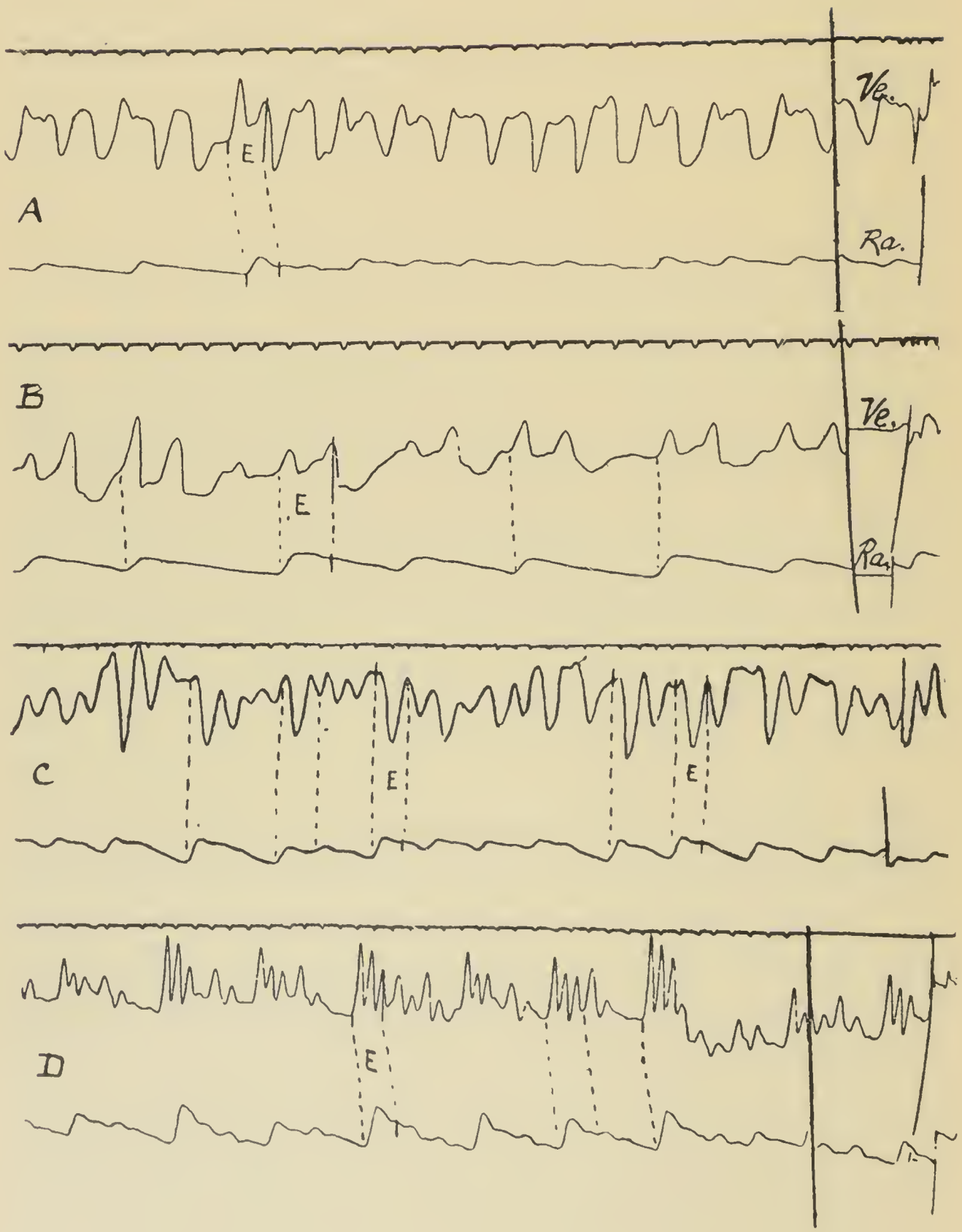
"The ventricular form of venous pulse" is a term which expresses the only fixed quality manifested by graphic records taken from the jugular veins in these cases. It implies that all prominent and rapid changes of volume in the venous cistern fall within the limits of ventricular systole. The curves corresponding to the individual heart beats vary in their positions relative to each other just as do the radial beats. There may be considerable variation in the amplitude of the separate curves in a given case. This variation is far from instrumental in origin, for close examination reveals the recurrence of a particular type of curve with a given length of pause, a given type of radial beat or a given phase of respiration. As a general rule and in a single case a large venous curve accompanies a large radial curve, but the difference in size from one beat to the next is less in the former than in the latter. A family resemblance between the separate venous beats of a single curve is generally if not always present.

The variation from case to case consists mainly in the relative difference in the height of the several waves and a similar difference in the depths of the depressions which separate them.

The complete curve, corresponding to a single heart cycle, is generally composed of two* or three peaks, and a similar number of dips. The upstroke of the first peak is synchronous with the commencing carotid pulsation at the same level of the neck (though it may precede or succeed it slightly). The downstroke of the last peak starts at a point corresponding to the opening of the tricuspid valves (Mackenzie). It is synchronous with the bottom of the downstroke of the cardiogram, or with a point a little later than the bottom of the dicrotic notch on the carotid tracing. The chief depressions follow the first and last peaks and are very variable in degree from case to case. As a general rule it may be said that the shorter the

* The waves were named by Mackenzie a^1 and v^1 respectively, and attributed to auricular and ventricular systole; de Vries[5] has recently shown that they are sometimes met with when the normal heart sequence is present. This accords with my own experience. I do not think that there is any type of the ventricular form of venous pulse which does not find its counterpart in the ventricular portion of the auricular form of venous pulse. It is probable also that with more extended observation the reverse will be found to hold, namely, that, in respect of their systolic elements, duplicates of all forms of the ventricular portion of the auricular form of venous pulse exist in the absence of all signs of the normal auricular contraction.

duration of the abnormal rhythm the deeper is the first as compared to the second depression; and that in old-standing cases the dip in the centre of systole is replaced by a larger and fuller complex of systolic peaks. A definite relationship appears to exist between the mean distension of the veins and the swelling of those veins in systole. Thus, in cases of long duration, in which the veins are more or less markedly dilated, the venous curve is in the form of a prominent systolic plateau. The older conception that the prominence of the venous pulsation is an index of the degree of tricuspid reflux is not without a definite foundation. The curves obtained from patients soon after the onset of the new rhythm and the curves in cases in which compensation is complete generally permit of close comparison with the *ventricular* portions of the venous curves taken from normal subjects (the parts of the normal curves usually marked *c* and *v*). A curve of the kind is shown in Fig. 2 C. In long standing cases, or in instances where compensation is less complete, the first depression (corresponding to the *x* and *x'* dips of the normal curve) is filled (Fig. 2 B and A), and the filling may happen in greater and greater degree until the type assumed is flat-topped and resembles the curve of intra-ventricular pressure (Figs. 2 A and 4 A). The transition from one type to the other may be followed from case to case, or may be seen in one and the same case as cardiac tone and venous filling wax and wane. The flat-topped type also consorts more commonly with rapid heart action, though in marked degree it rarely occurs in the absence of appreciable heart distension. In many curves the smaller and more rapid beats are accompanied by the flat type of curve, and the stronger beats by a bifurcated type (Fig. 2 A). The contrast may be explained by the unequal heart filling under the two conditions. The accompanying figure illustrates several of the types of curve met with, and others will be found in other parts of this paper. It is by no means unusual to see three prominent peaks, concurrent with each heart beat, and such curves bear a close resemblance to the experimental venous curves of Rihl[55]. The relationship of the normal type of curve to the various forms of ventricular venous pulse curve is diagrammatised in Fig. 3. The outlines have been drawn from actual curves, several of which illustrate this paper. The dotted outline is that of the auricular form of venous pulse. The essential difference between this diagram and the somewhat similar ones figured by Mackenzie[37] and Wenckebach[66] lies in the fact that it displays transitions between various types of the ventricular form of venous pulse. The original figures were published to illustrate the passage of the auricular form to the ventricular. The view of gradual "paralysis" of the auricle, which they were intended to express, and of the gradual distension of the chamber as a factor in the production of the true ventricular form of venous pulse and its concomitant, irregularity of the heart, has been entirely abandoned. It is perfectly true that dilatation of the auricle occurs, and it is equally true that such distension is accompanied by a transformation of the systolic portions of the venous curve as it has been illustrated. But the distension of the auricle and the deforma-


Ve.
E
A
Ra.
B
Ve.
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C
E
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D
E

Fig. 2. Simultaneous venous and radial curves from four patients with complete irregularity of the heart and the ventricular form of venous pulse. The figures illustrate some of the different types of venous curve met with in this condition. The venous curves are constant in that all rapid upstrokes lie within the confines of systole (E, E). The dotted lines in this and subsequent curves unite points representing one and the same phase of the cardiac cycle. The heavy vertical lines cutting a whole tracing mark the points where curve has been excised.

Curve A.

CASE 5.—R., a man aged 39. The mother had rheumatic fever. Admitted for shortness of breath and precordial distress. Heart enlarged right and left, veins prominent and liver dulness increased. No dropsy. Heart sounds weak but otherwise normal. Electrocardiograms typical, oscillations maximal over auricle. Four inches of curve have been excised from this figure.

Curve B.

CASE 6.—Mrs. K. P., aged 23, the subject of mitral stenosis of rheumatic origin. Admitted with enlarged liver and dropsy. The curves were taken some weeks after admission when the patient had responded well to digitalis. Early and mid-diastolic murmurs; electrocardiograms typical. Eight inches of curve excised.

Curve C.

CASE 7.—M., a man aged 26, with no symptoms other than slight short-windedness on strenuous exertion. History of rheumatic fever. No cardiac enlargement, no murmurs. Irregularity has been present for two years and has been persistent. The electrocardiograms show prominent oscillations and ventricular extrasystoles. (Fig. 24 is from this case.)

Curve D.

CASE 8.—Mrs. A., aged 38, suffering from mitral stenosis. On admission dropsy and œdema of lungs were present. Tracing taken several months later, subsequent to digitalis. Compensation had so far improved as to allow her to pursue her ordinary duties as housewife. Heart enlarged to right and left; early and mid-diastolic murmurs. Veins not prominent. Irregularity persistent. Two inches of curve excised.

tion of the venous curve may take place, while, during the transition, the presystolic auricular contraction is present, or while, during the transition, the co-ordinate systoles of the auricle are suspended. As we shall see at a later stage the ventricular form of venous pulse may be conspicuous even in its plateau form, while the normal heart sequence is maintained (*CASE 14*), and, as we have already seen, the usual or normal systolic portion of the venous pulse may be found (the type with the deep x and x' depressions) and yet the signs of co-ordinate and presystolic auricular contraction may be entirely in abeyance. It should be clearly comprehended that two

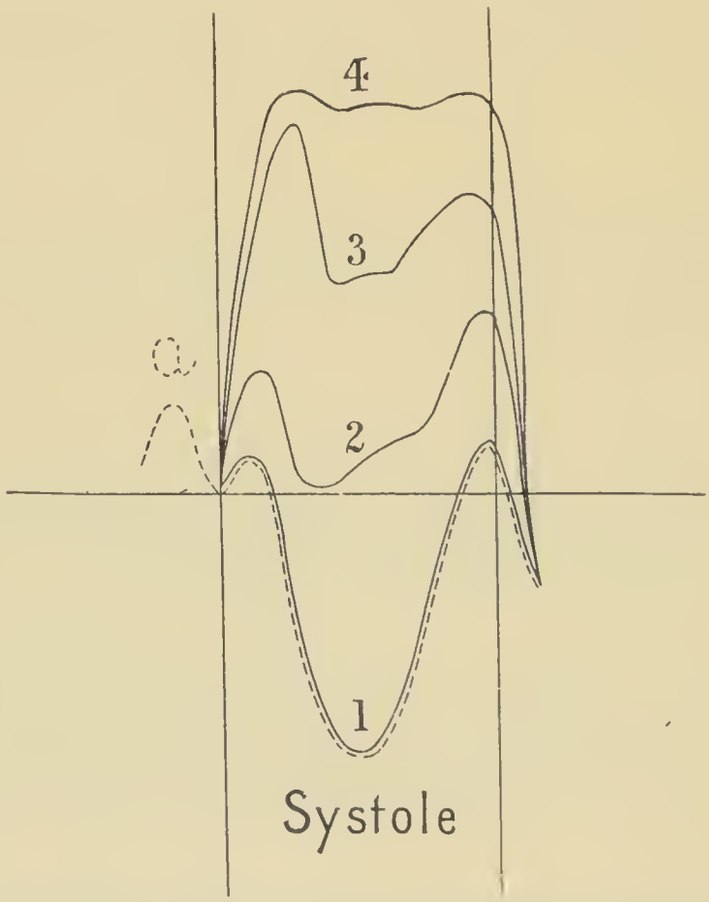

Fig. 3. A diagram illustrating the relationship of the auricular and ventricular forms of venous pulse, and the more common variations in shape to which they are liable. The dotted line represents the usual or physiologic type of the auricular form of venous pulse. The continuous lines represent the ventricular portions of types of curve met with in auricular and ventricular forms of venous pulse.

perfectly distinct phenomena exist, a sudden disappearance of all signs of the normal auricular systole, and a gradual increase of pressure in the right side of the heart. The lack of due appreciation of this essential distinction has led in the past to many misconceptions of the conditions with which we are dealing, and to the faulty interpretation of many curves. The diagram may stand as an example of the transition of the systolic portion of either

auricular or ventricular form of venous pulse curve, from the compensated to the decompensated type, or from the type with perfect to the type with imperfect venous flow. In brief, it may be said that there is but one distinctive quality between auricular and ventricular forms of venous pulse, and it consists in the absence of the auriculo-systolic or *a* wave from the former. Individual cycles of a curve, which is an example of the ventricular form of venous pulse, can be identified as such with certainty only by attention to this fact.

These considerations bring us to a subject of importance to those for whom the application of graphic methods is difficult or impracticable. The question may be asked—Can the ventricular form of venous pulse be recognised with the unaided senses ? The reply to this question is that it can be recognised in a very large number, in fact, the majority of cases, if note is taken of the conjoined phenomena. The first essential in its recognition is the finding of an irregular pulse. If the heart is beating arrhythmically the ventricular venous pulse may be identified in almost all cases in which it is prominent. Each beat of the heart as felt at the apex is accompanied by a pulsation in the veins of the neck, which starts with the systole and preserves this relationship from beat to beat. Now the prominence of venous pulsation is very variable. It may be so marked that at each systole the girth of the neck is increased by an inch or more. The pulsation commonly attains the angle of the jaw, and not infrequently it displaces the lobe of the ear. In one case I have seen it extending as far as the summit of the scalp and have recorded it above the zygoma. But a type so marked is exceptional. Much oftener the pulsation is confined to the neck. If, under these circumstances, the superficial veins are conspicuous, it is readily recognised. But in cases where this is not the case I have seen it mistaken for arterial pulsation on several occasions. The error may be avoided if it is borne in mind that the pulsation is a volume change and not a pressure wave ; it is often visible, but the fingers rarely appreciate it. "Diastolic collapse" often accompanies it, and is merely a descriptive term for the impression left on the sight by the rapid volume decrease which occurs at the end of systole. This volume decrease is shown in the curves, and has been referred to previously. In early and compensated cases the pulsation is rarely recognised by the ordinary means, for usually in such instances the visible pulsation is complex ; resort must then be had to the graphic method. A venous tracing may often be secured where no venous pulsation can be seen.

Certain waves which occur in diastole.

It has been stated that the ventricular form of venous pulse is composed of waves of which the prominent and rapid ones fall during systole. It is also true that waves occur within the limits of diastole. The earlier interpretations of the curves in which they are found are, almost without exception,

erroneous. The waves in question have been attributed repeatedly to co-ordinate contraction of an irregularly beating auricle (in a recent paper Magnus-Alsleben has fallen into similar error)[42].

The view has been expressed that apart from the auriculo-systolic wave there is no essential distinction between the individual venous curves in the normal and abnormal conditions*. It will be necessary at a slightly later stage to note a single exception to this rule. But, for the time being, we may enquire whether any waves occur in the diastolic reaches of the normal venous pulse, apart from those allotted to auricular systole. There are waves of two kinds, and precisely similar waves are found during diastole in the condition which we are considering. There is a wave which has been termed by Morrow[16] the "second onflow wave," and its eausation is assigned to the overflow into the veins when in a long diastole the heart is already full, or to the increase of pressure in the ventricle during the filling. It is seen to advantage in the accompanying figure (Fig. 4). At the commencement of tracing A there is a long pause, and during the whole of it the veins are steadily swelling. It is also prominent in tracing C and is shown to a lesser extent in tracing B. The fixed relationship of this stasis wave to the preceding systole and the variation of its extent with the length of diastole permit its recognition.†

The second type of wave was first described in the normal eondition by Hirschfelder[23], and has been attributed to the closure of the tricuspid valves at the end of ventricular filling (Gibson[10]). But there is evident misconception as to the extent to which this interpretation can be applied. The snapping to of the auriculo-ventricular valves will not serve in explanation of a permanent increase in venous volume. The type of wave produced must be carefully distinguished from that produced by stasis. If the wave is followed by a dip it may perhaps be assigned to a valve movement, but only, I think, such part of the wave as is definitely raised above, or depressed below, the general sweep of the stasis wave itself. Waves belonging to this class are seen in Fig. 4 B and C. In tracing C, and just at the summit of the main sweep of the stasis wave a triple vibration (marked ×) with constant time relationship to the preceding systole is plainly visible. It was accompanied by a distant valvular sound at the apex and base of the heart (cp. Gibson[10] on third heart sound), and by a distinet double clieking sound in the veins of the neck. In tracing B we have an example in which the portions of the diastolic waves attributable to stasis and valve movement are more difficult to unravel. The longest diastole, just antecedent to the stops, is of value in this connection. In this diastole we see the general sweep of the stasis wave and superimposed upon the first part of it is the wave marked ×. The same wave is found in earlier portions of the same

* The statement is not affected by the fact that one type of ventricular curve is more common in the normal and another type in the abnormal condition.

† Its inclination to the vertical is a rough index of the rate of venous flow.

curve, and the comparison with the later diastole allows us to gauge the extent to which stasis aids in its production in these earlier diastoles.

We may now turn to another type of diastolic wave, the most important of all. It forms the exception to the remarks previously made as to the resemblance between the ventricular and auricular types of venous curve. These undulations are only met with as an accompaniment of the ventricular form of venous pulse. They are seen but occasionally, and only when the pulse is slow (Wenckebach[65]). They are well marked in tracing A, and are more obscurely figured in tracing B (marked *f*). They are also shown in the photographic curve (Fig. 19). They are multiple, and the rate may be from 350 to 500 per minute. Their irregularity renders it difficult to estimate the rate other than approximately. They were first described by Wenckebach[65] in 1907; in discussing Cushny and Edmund's paper on auricular fibrillation and the relationship of the latter to complete irregularity, he stated that in the clinical condition he had seen small venous waves which might be attributed to small contractions in the auricular walls. Later, in the same year, they were figured by Mackenzie[37], who also suggested that they might arise as a result of auricular fibrillation. The relationship of this fibrillation to the view adopted in his papers, namely, the nodal origin of the rhythm, is not explained, and in a later publication[38] (p. 299) the idea was abandoned. ("From this observation, I now recognise that what I had taken for waves due to fibrillation of the auricle were really due to a fault in the method of registration, wherein by compressing the vein with the receiver I had artificially produced thrills which appeared as waves in the tracing.") Certain observations, which will be discussed more fully at a later stage, point to the conclusion that the oscillations are in reality the outcome of fibrillary movements in the auricle. At the present time it will be sufficient if I state that the thrills in question have not come under my personal observation, that I am inclined to attribute the thrill in this instance to the oscillations rather than the undulations to the thrill, and that in chlorotic cases where the auricular type of curve is obtained to perfection such vibrations are absent.

The electrocardiographic curves obtained by leads from the right arm and left leg.

Electrocardiographic curves from cases of complete irregularity of the heart have been published by Einthoven[6] (Figs. 17 and 31), and later by Kraus and Nicolai[26] (Fig. 14), Hering[18], and Rothberger and Winterberg[56].

The following account is based upon curves taken from thirty cases.

Firstly, the curves consist of tall peaks R, corresponding to the commencement of ventricular systole, and scattered throughout in irregular profusion. Their arrangement is obviously due to the arrhythmic action of the ventricle (cp. Fig. 15 and its accompanying radial curve). The direction of the electric variation, of which the peak R is an expression, indicates

negativity of the arm electrode, or primary activity of the base of the heart. The variation is the same as that which occurs in the normal subject and is an evidence, as Hering has pointed out, that in this condition the ventricular contraction commences at its normal starting point. The remainder of the curve and its comparison with the normal (normal curves are shown in Figs. 12 and 16) justifies this conclusion, as we shall see in the sequel. I have long suspected that the peaks R in the abnormal curves are relatively higher than in the normal curves. For curves of good excursion are always obtained with facility even while the instrument is adjusted at a comparatively insensitive point and in patients with high resistances. The comparison may be made in Figs. 15 and 16, taken from the same patient within two days of each other; it may also be made in Fig. 9 (it is mentioned in the explanatory remarks attached to the figure*).

A comparison of the height of the peaks R with the strength of corresponding radial beats in synchronous tracings reveals the fact that there is no fixed proportion between them (Fig. 15). A very similar, if not identical, phenomenon is present in the condition known as heart alternans (for the experimental fact, see Hering[18]; the clinical parallel has been observed more recently (*CASE 15*)).

Apart from the peaks R the normal ventricular curve consists of slight depressions following them, and these in turn are succeeded by broad waves designated T. The T variations are also seen in curves from cases of complete irregularity. They are often very obscure, and this obscurity is the result of the presence of certain special oscillations upon the curves. The variation T is plainly perceptible in Figs. 13 *I*, 18 *I* and 19, while it is overshadowed in Figs. 14 *I*, 15 and 23. At times it is quite as prominent and almost as regular from cycle to cycle as in the normal curves (Fig. 19). The occurrence of T is of importance because it demonstrates, what would otherwise be uncertain, that the ventricular contraction starting at the base pursues its usual path in the ventricle†.

The most striking feature of the electric curves is the absence of all sign of the regular presystolic variation, which accompanies all normal heart beats (P in Fig. 16), and the presence of that which replaces it, namely a number of irregular oscillations varying in form and prominence. In leads from arm and leg these oscillations are often marked in amplitude, and the latter may equal if it does not exceed that of the usual auricular variation P (Fig. 23). In other curves they may be far less conspicuous, and may be

* An increase in the size of the peak R as compared with the normal was also seen in the cases of regular paroxysmal tachycardia which have already been placed on record[30 & 33]. It would appear that both in complete irregularity and in the regular paroxysms the heart, as a whole, is in a state of hyperirritability. The curves seem to indicate at all events that in such cases the energy developed and used up is great in proportion to the work accomplished. In other words, the heart is working at a disadvantage. Einthoven[6] found no increase, but in one case a decrease, in the size of R with an increase of rate following exercise (Figs. 26 to 29 of his paper).

† The variation T is attributed by Gotch[12 & 13] to a return of the contraction to the base of the heart (activity is represented electrically by negativity).

distinguishable only from place to place (Fig. 24, *ff*). In one form or another they have been invariably present in all the cases examined. They are a constant feature of cases of complete irregularity of the heart and occur in no other condition. They are responsible for the distortion or concealment of T in the arm-leg leads ; the clean cut character of R is never affected, and this is due to the quickness of the movement. The oscillations appear in a purer form when certain special leads are adopted, but the curves obtained in this way will be more appropriately dealt with in a subsequent section.

The oscillations are most conspicuous when the pulse is slow or during a diastole of unusual length. For during systole they fall upon the ventricular elements of the curve and their definition is obscured. When the heart beat is rapid the individual waves may be difficult to separate, but their presence is known from the fact that the T waves vary greatly in form or are entirely overshadowed (Fig. 15), and by the appearance here and there of single undulations on the curve which can only be ascribed to the same cause. The deformity of the curve as a whole, produced in this manner, is so characteristic of the condition that, once recognised, the curves are never mistaken for those obtained in any other affection.

Complete irregularity of the heart is the result of auricular fibrillation.

The irregular oscillations seen upon galvanometric curves are due to an incoordinate contraction of some portion of the heart ; they are not a direct result of structural change in the heart, and are independent of movements of the somatic musculature.

It might be suggested that the oscillations in question are dependent upon structural changes in the heart muscle, and that the propagation of the contraction wave, along the path usually taken, is hindered or modified by the presence of areas of tissue affected by disease. But we are in a position to deny that the oscillations are attributable to this cause.

Such a hypothesis is at once weakened when it is known that the oscillations bear no relationship in their extent or frequency to the objective signs of damage in the myocardium. For while a patient may be suffering from obvious and gross myocardial change, yet if the sinus rhythm is dominant the oscillations are absent ; and, on the other hand, patients showing no signs of gross myocardial affection, patients in whom there is no dilatation and in whom there is little disturbance of the circulation even after strenuous exercise exhibit fully developed oscillations, provided that the pulse is completely irregular. If further evidence is required it will be

found in two cases in which a comparison of the normal and abnormal mechanisms in the same subject was secured*.

CASE 10.—G. P., aged 77, was admitted under the care of Dr. Mackenzie, to whom I am indebted for opportunities of examining him, in September, 1909. There was no history of past illness, and rheumatism and chorea were unknown in the family. He complained of slight cough and expectoration, and huskiness of voice of four months' duration. Anginal symptoms had been present. There had been no shortness of breath, palpitation, giddiness or dysphagia.

Condition, 22-10-09.—A strongly built man; the face weather-beaten. The voice is husky, laryngeal examination shows abductor paralysis. A slight grade of cyanosis is present. The arteries are thickened, the pulse is completely irregular; the venous pulse is of the ventricular form. The heart's apex is obscured by emphysema. The right line of dulness is ½ inch and the left 3½ inches from the middle line. There is an area of dulness extending into the second left space and at this point systolic pulsation was present upon admission. But for the irregularity the heart sounds are normal. The urine is normal.

The case may be summed up as probably one of aneurism of the thoracic aorta, associated with angina and complete irregularity of the heart. Subsequent to his admission he has had many anginal attacks, some of a severe grade. There have been times when the pulse is slow, and on such occasions it is regular and the venous pulse has been of the auricular form, and times when it is fast and irregular, the venous pulse being then ventricular in outline. The paroxysms of irregularity have been numerous, and they have generally lasted for 24 hours or more. On several occasions the pulse has been irregular for several days together. On the other hand attacks of comparatively short duration have occurred. There have been no definite symptoms at the onset or offset of attacks, and the patient has been unaware of the abnormal cardiac mechanism when it has been present. The anginal attacks have had no relation to the periods of irregularity.

Two of the curves (Figs. 15 and 16) obtained from this patient were taken within 48 hours of each other. Upon the day when the first electrocardiogram was obtained polygraph curves showed the presence of complete irregularity and the ventricular form of venous pulse. The electric curve corresponding (Fig. 15) is of the form which we have been considering. The peaks R are separated by stretches of curve of an irregular character. No two pieces are alike, and the T variations are obscured by the oscillations which are present. Two days later the pulse was regular and the jugular curve demonstrated a prominent *a* wave. The corresponding electrocardiogram is given in Fig. 16. In it the disappearance of the oscillations is associated with the return of the auricular variation P, while the remainder of the ventricular curve is of a perfectly normal type. Curves showing the normal rhythm within half an hour of the cessation of an attack have since been obtained from this patient. They were of a perfectly normal type. Digitalis, given in doses sufficient to produce toxic effects, had no retarding influence during the periods of irregular tachycardia.

* A third case will be found in a later section and the curves are illustrated by Fig. 9.

CASE 11.—A. G., a barman, aged 24, was admitted to the out-patient department at the City of London Hospital on 8-12-09, complaining of cough with blood-stained sputum, and pain in the left shoulder. These symptoms had been experienced for some months; but he has had earlier attacks and is known to have suffered from heart disease for several years. He has never had rheumatic fever or chorea and there is no history of either in the family. He has never had dropsy.

Condition, 8-12-09.—The right limit of cardiac dulness is 2 inches, and the left 5 inches, from the middle line. There is a marked presystolic thrill at the apex; a rough crescendo murmur leads up to an accentuated first sound. The second pulmonic and aortic sounds are normal. There are no signs of liver enlargement or dropsy, but there are scattered rhonchi over both lungs. Auricular extrasystoles are frequent and interrupt an otherwise regular heart rhythm. Each extrasystole is accompanied by a first and usually by a second sound, and a dull valvular sound precedes the first sound, but there is no murmur. The *a-c* interval is increased. A polygraph curve is shown in Fig. 5*a* and electrocardiograms in Fig. 12. 13-12-09.—On this day the patient's condition was identical with that already described. He was placed on tincture of digitalis mX t.d.s.. 18-12-09.—The patient awoke in the early morning (he has remained in bed since he first attended) feeling ill. He experienced violent palpitation accompanied by a choking sensation in the throat and difficulty in breathing. He states that his heart was beating rapidly and irregularly. Brandy gave him relief but the irregular action of the heart remained. The attack lasted ¼ hour. He has never had palpitation before which in any way resembled it. 20-12-09.—On examination the right limit of cardiac dulness is 1½ inches and the left 4½ inches from the middle line. The heart is absolutely irregular, and the venous pulse is of the ventricular form. The auriculo-systolic murmur is no longer present, but is replaced by a murmur of very similar quality which occupies early diastole and continues into the mid-diastole of the longer cycles. The murmur consequently fills the diastole of the shorter cycles. The polygraph curve is shown in Fig. 5*b* and electrocardiographic curves in Fig. 13. The digitalis was omitted on 20-12-09, but was readministered in larger doses at a later date. The pulse subsequently slowed down in the characteristic fashion, dropping at times to 40 and 45. The digitalis was then relinquished, and the pulse rate returned to 90-120.

Briefly, the case is one of old standing mitral stenosis coming to hospital for an attack of hæmoptysis. Within ten days of attending (and possibly as a result of the digitalis) a normal rhythm interrupted by auricular extrasystoles gave place to complete irregularity of the heart. This irregularity has been present ever since.

Fig. 5*a* is a polygraph curve taken from this patient on 8-12-09. It shows the normal sequence of chamber contraction, but the *a-c* interval is increased to 0·28 sec.. The rhythm is interrupted by frequent auricular extrasystoles (the premature auricular waves are marked *a'* in the curve) of which three examples are shown. The electrocardiographic curves taken upon the same day are exemplified by Fig. 12. The upper curve of this figure consists of parts of four normal contractions and of two auricular extrasystoles. Reading from left to right, we see a normal beat, accompanied by P, R and T variations; this is succeeded by a second beat of the same character except that the T variation has a premature P wave superimposed upon it; its height is consequently exaggerated. The premature P variation is followed by the R and T peaks of the premature ventricular systole, and the latter is followed by a pause which is not fully compensatory. A normal contraction, an auricular extrasystole and a portion of a normal contraction complete the curve. In the lower curve similar events are shown. Here three contractions are followed by a pair of extrasystolic beats arising

in the auricle, and the curve ends with a normal contraction following a short pause. The features of these curves to which attention is directed are several. The P-R intervals of the normal beats is increased, amounting to a full 0·2 sec.. The P-R interval of the extrasystoles is difficult to measure, but certainly exceeds this fraction. The curve is clean cut and shows no trace of oscillation.

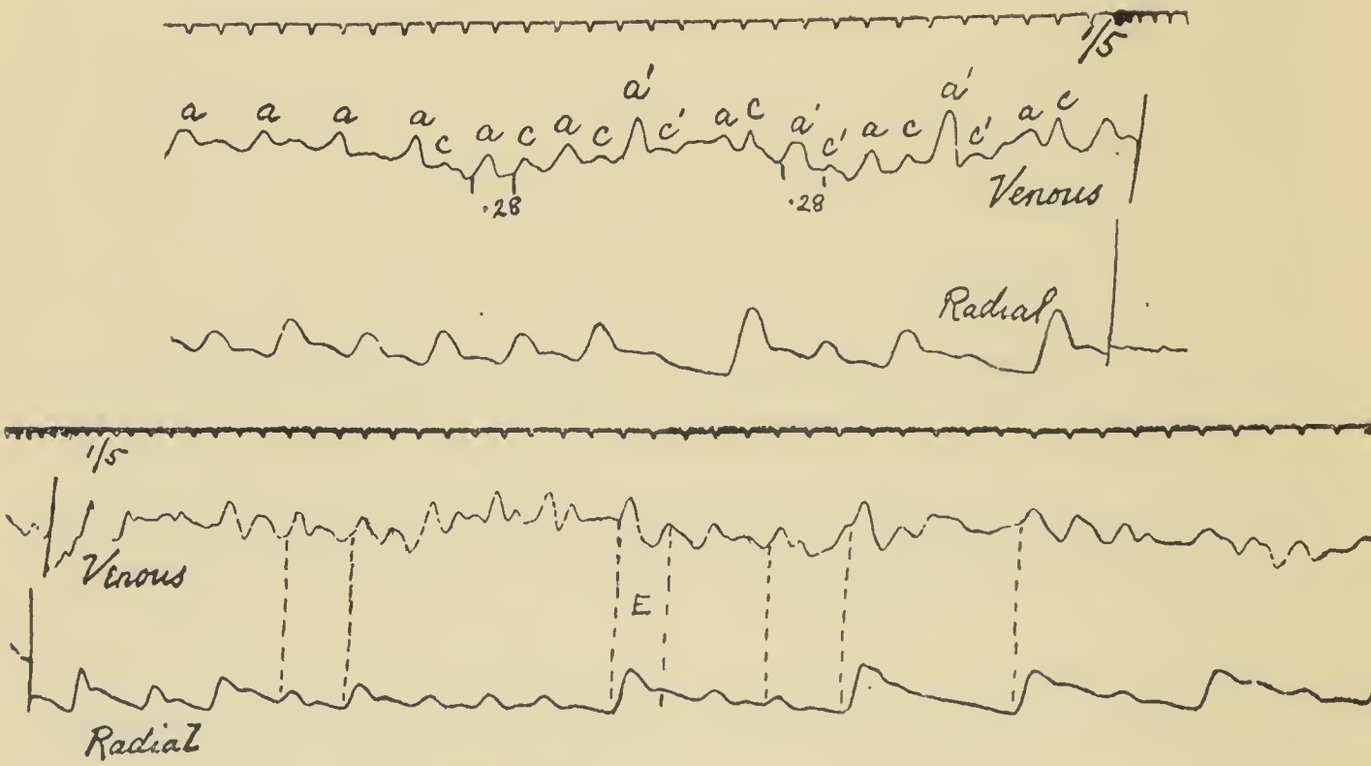

Fig. 5 *a* and *b*. Two polygraph curves obtained from *CASE* 11, on 8-12-09 and 20-12-09 respectively. Tracing *a* shows a venous curve of the auricular form, and auricular extrasystoles are present. The *a*-*c* intervals are increased. Tracing *b* shows a curve of the ventricular type, the pulse is completely irregular.

Fig. 5*b* illustrates the condition present two days after the onset of the complete irregularity and twelve days later than the occasion on which the preceding tracings were secured. The polygraph curve shows complete irregularity of the arterial pulse, and the ventricular form of venous pulse. The electrocardiogram, taken on the same day, is given in Fig. 13 *I* (Fig. 13 *II*-*V* will be discussed later). The curve is characteristic of the condition, and is composed of peaks R, the remains of peaks T, and numerous irregular oscillations. There is no sign of the normal P variation. The dilatation of the heart had distinctly diminished, and the general condition had improved, at the time when the second group of curves were taken.

Now as it is impossible to allow either in the first case or in the second that there was any material difference in the condition of the heart muscle during the brief intervals which intervened between the occurrence of the

normal and abnormal types of curve, for in both patients the cardiac affection was essentially a chronic one, so it is equally impossible to agree that the oscillations displayed were directly dependent upon myocardial change. On the other hand the cases provide very suggestive evidence that we are dealing with a temporary ineo-ordinate action of a limited portion of the heart muscle ; and as the evidence of normal activity in the auricle is absent it is essentially towards that chamber that our attention should direct itself. The cases demonstrate conclusively the interdependence of the abnormal type of heart curve and the gross irregularity. Further, they afford a strong argument against the view adopted by Hering[18] that the oscillations have their origin in the somatic musculature. The normal and abnormal curves were obtained under precisely similar conditions.

It is perfectly true that many electrocardiograms show traces of variations produced by contraction of the muscles of the body wall or limbs, and at times it may be difficult to exclude this complication from the curves. But the oscillations of which we are speaking bear no relationship to the extent of such movements. Muscular movements give rise to irregularities in the curves when a patient trembles or fidgets. In the great majority of such cases these extraneous vibrations can be identified at once by their general appearance and rate. If precautions are taken in the avoidance of elderly or tremulous subjects, if the recumbent posture is adopted, and if in a warm room absolute stillness is enjoined and enforced, no such irregularities appear in subjects in which the heart sequence is normal. Oscillations are invariably present in the class of patient considered, whatever the precautions employed. They are of much the same degree from day to day and from hour to hour in the same subject. They are equally prominent when leads from the two arms are adopted, but vanish almost completely if the electrodes are attached to the two inferior extremities. The proposition, that they are part and parcel of the heart beat as it is represented to us electrocardiographically, is unequivocal. Very numerous and special leads have been devised and employed for the exclusion of their origin in abdomen, limbs and head and neck. The special leads show that it is a matter of indifference, so far as the amplitude of the oscillations is concerned, as to how great is the extent of somatic musculature which lies beneath and between the electrodes. They demonstrate that the excursion is controlled by the proximity of the heart to the leads.

The special leads take us a step further, for they make it clear that it depends upon the part of the heart approached as to how conspicuously the oscillations will appear.

The irregular oscillations arise in the vicinity of the auricle ; the ventricular electric complex in complete irregularity is of the normal form.

Special electrodes were employed, composed of small circular copper plates, and these were fixed to the chest wall by means of a layer of stiff

flour paste made up with a large addition of common salt. A great variety of curves can be obtained in the normal subject by the adoption of the many leads which offer themselves, but with these we are not at present concerned ; our attention may be confined for the time being to the amplitude of the oscillations which occur in patients who manifest complete irregularity of the heart. The oscillations are never seen and no vibrations are found, which in any way resemble them, in normal subjects or in patients who exhibit the normal sequential cardiac mechanism. But in cases where the pulse is irregular and the ventricular form of venous pulse is present they are invariable. The analysis, which the chest leads afford, is provided most strikingly by patients in whom the right auricle is enlarged, for in these a larger area of the auricular wall is in apposition to the chest wall; but the phenomena which are here described are not limited to such cases, though present in them in greater degree.

Fig. 13 shows five separate leads. *I* is from the right arm and left leg, and has been mentioned already. The remainder, *II-V*, are from the chest wall and were taken at the same time and under similar general conditions. *II* is the curve yielded by electrodes, one (the arm electrode) placed upon the sternum at the level of junction of the second rib, the other (or leg electrode) upon the apex. The ventricular complex is represented by a curve similar to that often obtained in arm and leg leads from normal subjects ; oscillations are also seen on the curve, but they are not prominent except towards its termination. *III* was obtained by moving the apex electrode to the fourth space on the right side and one inch from the sternal margin. The ventricular peaks are comparatively small, and the oscillations are maximal. The leg electrode was next replaced at the apex and the arm electrode was fixed, first in the third space in the anterior axillary line on the left side (*IV*) and later, just below the costal margin three inches internal to the apex beat (*V*). The curves may be taken as representing in the main the pictures yielded by left and right ventricles respectively. They are almost entirely free of oscillations, and show regular and clean cut variations in the latter part of each systole.

The separate leads which we have examined, and numerous leads from other parts of the chest wall, demonstrate at the outset that the oscillations are conspicuous or the reverse according to the proximity or otherwise of electrodes and right or superficial auricle. The conclusion is obvious and beyond question. Of the area of heart superficies which is in relationship to the front of the chest wall it is only over that portion of it which may be termed auricular that the maximal oscillations are produced, and as the heart is admitted as the source of the oscillations their origin may be traced to the auricular portion of it.

Separate leads from the chest wall have been obtained in a large number of patients with the irregularity in question, and they all yield similar results. Two further examples are given in Figs. 14 and 18. The explanations of the figures may be consulted for the details of the leads.

The special curves give an analysis of those obtained by means of the usual arm-leg leads, and show that the latter are constructed by the superimposition of current curves of auricle and ventricle. They explain the deformation of the variation T and show that the variability of this peak from cycle to cycle is the result of the combination of the currents derived from the two sources, and that it is not an anomaly of the ventricular complex itself.

Those curves which are obtained by means of leads from the area overlying the auricle give the purest pictures of the oscillations which may be gained in the human subject (it is possible that œsophageal leads would give still more distinctive tracings) and allow a more detailed study of the oscillations.* Obtained in this way they are found to vary in rate between 400 to 600 per minute, but the usual rate approaches 500 very closely. They frequently have a character, which, though at present inexplicable, is remarkable, and it is seen to advantage in Fig. 13, *III*. The individual oscillations rise abruptly and fall away more gradually. It is a trait not infrequently manifested by the undulations found with arm and leg leads, but in these it is seldom so clearly defined.

Certain deductions from the clinical findings.

The outstanding feature of the records from cases of complete irregularity of the heart is the entire absence of all sign of the normal auricular systole. It has been shown time and again that, while in normal subjects and in patients suffering from all other forms of heart irregularity or cardiac disability the auricular systole leaves a definite impress either upon the cardiogram, upon the venous volume curve, upon the œsophageal curve (Minkowski[45], Rautenberg[51], Young and Hewlett[68] and other writers), or upon the electrocardiographic curve, such evidences of its normal activity are consistently wanting by each and all of the graphic methods in the group of cases which is engaging our special attention[52].

The conclusion which it is impossible to avoid, a conclusion which is accepted by all those who have given reasonable attention to the subject, is that the normal presystolic auricular contraction is in abeyance, temporarily or permanently.

Paralysis of the auricle has been suggested, but little support is now found for this hypothesis. The conditions of the circulation are often such that it is impossible to suppose that the pressure in the auricles is increased. Moreover it is dubious whether we are justified in applying the term paralysis to heart muscle, if we mean by paralysis complete inactivity of the muscle. The maintenance of a state of complete flaccidity in cardiac muscle, surrounded by conditions favourable to its nourishment and to its contraction,

* Very large oscillations may be obtained by placing one electrode over the right auricle in front and the other in the neighbourhood of the angle of the right scapula.

for an appreciable length of time, is a proposition foreign to the experience of experimentalists. A deprivation of its functional powers would lead us to expect, from the analogies provided by pathology, an obliteration of its macroscopic and microscopic characteristics. The observation of hypertrophy in the auricle at autopsy, led Mackenzie to abandon his earlier view of paralysis and influenced him in concluding that the auricle is active. The view was supported by the reappearance of signs of auricular contraction in paroxysmal cases. We are led to a precisely similar contention by the evidence yielded by the electrocardiographic curves. The auricle is the seat of an electric disturbance of a peculiar yet distinctive nature. The constancy of the oscillations, their unique appearance and *their presence throughout the whole of the cardiac cycle*, is responsible for the convictions that they are an essential feature of complete irregularity and that *the activity of the auricle is continual.*

Co-ordinate contraction of the auricle at any period of the cardiac cycle other than that of ventricular systole can be readily excluded. Co-ordinate and simultaneous contraction of auricle and ventricle can also be set aside, for, as will be stated in the sequel, it gives rise to an entirely different clinical picture (see p. 360).

As a whole, the evidence points in the most convincing manner to the conclusion that co-ordinate contraction of the auricle is absent, and to the conclusions that activity is present, and that this activity is a continuous one.

In experimental work we encounter but one variety of auricular activity in which inco-ordination of the separate fibres is present, and this mechanism is one in which the auricle is in unceasing movement. It is the state known as fibrillation or delirium.

The remainder of this communication, therefore, in so far as it deals with the mechanism of the irregularity, will be directed in the main to a comparison, of as searching a nature as possible, between complete irregularity in man and auricular fibrillation as it is induced in the lower animals. For it is upon this comparison that the survival or overthrow of the hypothesis suggested, namely the identity of the two conditions, must ultimately depend.

Oscillations similar to those characterising complete irregularity of the heart in man are found experimentally when the auricle is fibrillating, and these oscillations are produced in the auricle as a result of the fibrillation.

For the purpose of experiment dogs have been employed. They have been rendered insensitive with morphia and paraldehyde, and during the course of the experiments complete or deep surgical anæsthesia has been maintained by the administration of a sufficiency of ether. Fibrillation of the auricle has been induced by faradic stimulation of an auricular appendix. The auricle was exposed by one of two operative measures, by making a

window in the chest wall, or by splitting the sternum and exposing the whole heart. Utilising the former route a small incision has been made in the pericardium and, two fine insulated wires being attached to the auricle, the opening in pericardium and chest wall has been closed. By withdrawing the air from the chest the natural conformity of the chest and spontaneous respiration could be restored. The second method has been confined to experiments in which it was necessary to obtain synchronous myocardiograms from auricle and ventricle or to those in which electrocardiograms taken by means of direct leads from the heart were desired. Simultaneous arterial curves were obtained with Hürthle's manometer, simultaneous venous curves by employing portions of the polygraphic apparatus.

The electrodes adopted for the galvanometric leads were of two kinds. In leading from the right fore-paw and left hind-paw the clinical electrodes were utilised. In leading direct from the heart the flexible electrodes described by Gotch[13] were found to be most serviceable. The auricle was faradised until fibrillation was established, the stimulation was withdrawn and the curves taken before, during or after the return of the normal or sequential contraction.

The oscillations which are obtained experimentally as a result of faradic stimulation of the auricles are unique, for they occur in no other experimental condition. They are seen in Figs. 20 and 22. They consist of variations which succeed each other rapidly, at a rate, in the experience of these experiments, varying approximately from 500 to 900 per minute.* They replace the usual P variations of the normal rhythm and produce the same deformation of the T variations as in the clinical curves. In leading from the terminations of superior and inferior vena cava, the oscillation is sometimes characterised, as is the clinical oscillation, by its more abrupt rise and more gradual fall (Fig. 31 *II*).

Though never absolutely regular, yet at times the spacing is remarkable for its tendency to regularity, a quality noted in the clinical curves. They continue throughout the whole of the cardiac cycle.

The proposition that these variations are generated in the auricle and that they depend upon the delirium in the walls of that chamber is easily substantiated. They are only present when the auricle fibrillates, and are entirely absent from the curves yielded by the same animals when the normal rhythm is re-established. Figs. 20 and 21 are two simultaneous electrocardiograms taken from a single animal within a few minutes of each other. The first, during an auricular fibrillation period, exhibits the oscillations ; the second, after the re-establishment of the normal sequence, shows none, but the P variations have reappeared.

There is a relationship between the rate and amplitude of the electric oscillations, and the vibrations seen in myocardiograph curves. It is well

* The estimation of the rate of oscillations is necessarily only approximate, for they are irregular. The rates were calculated where several similar oscillations succeeded each other and the finest vibrations have been neglected.

known that auricular fibrillation may be of many grades, the movement of muscle levers is fine or coarse in the same experiment or from animal to animal. It is only rarely that an actual correspondence can be found between the individual muscle movements and the electric variations, and this is to be expected, for the whole auricle is active, and only a small portion of the muscular movement is recorded. Nevertheless, correspondence may be found in some degree. When the muscular movement is small, and the vibration of the lever rapid, the oscillations tend to be of small excursion and, as seen under vagal stimulation, may reach a rate of 900 or more per minute.* With the coarse fibrillary movements on the other hand, the electrical variations are of greater amplitude and are slower. Fig. 29 shows simultaneous curves from ventricle and auricle, and an electrocardiogram. In the left hand portion of the figure the auricle is fibrillating, the ventricular beats are irregular, the movement in the auricle is of the coarse variety. In this instance the auricular muscle, though fibrillating, appears throughout to be on the borderland of a return to co-ordinate contraction. Thus in the last half of the fibrillary period the separate vibrations, as seen in the muscle curve, are almost though not absolutely regular at a rate of 450 per minute. The movement in the first half of the fibrillary period is more disorderly and a little more rapid. The figure is a reduction of a greatly magnified photograph of the original curve. During the whole of the fibrillary period it is possible to establish a definite relationship between the individual movements expressed by the auricular curve and the oscillations which appear upon the electric curve. During the last half of the fibrillary period (R^7 to R^{12}) an oscillation falls with each of the peaks R and with each of the variations T. As a result the height of these waves is enhanced, and a picture bearing a superficial resemblance to 2 : 1 heart-block is produced. The rate of the auricular movement, the absence of absolute regularity and the great reduction of the auricular muscular movement when compared to the normal, demonstrate, however, that this portion of the curve cannot be interpreted in this way. In the earlier portion of the curve (beats 2-7) the auricular ineo-ordination is more clearly visible, but here also a complete analysis of the electrocardiogram is possible by exact fixation of the instants at which the individual auricular and ventricular movements fall. The curve as a whole is a complex picture composed of an accurate super-imposition of R and T variations, the result of ventricular systoles, and of auricular variations, marked P^1 to P^{18}. It would seem that the oscillation appearing in auricular fibrillation is the total result of the electric changes occurring in the individual fibres, and that similarly the movement of the myocardiograph lever is the total result of the combination of contractions and relaxations of the individual fibres. If at any given time a greater number of fibres are in contraction than there are fibres in relaxation, and such a state would be anticipated when the auricle is tending to return to a normal or co-ordinate

* Speaking of the coarser oscillations only.

mechanism, then a tendency to general contraction of the chamber as a whole, and an inclination for the electric variations of given direction to superimpose and produce a more or less prominent resultant curve would be present. There is evidence of a similar kind (which has been referred to in a former communication) in the case of fibrillation of the ventricle and the similar oscillations which are the outcome of it.

The location of the electric change in the auricle is a simple matter if direct leads are taken from the heart itself, and from various parts of the body wall. The oscillations are maximal when the lead is from the auricular substance (Fig. 31 *I* and *II*), and their amplitude decreases according as the distance separating auricle and electrode is widened (Fig. 31 *V* and *VI*). Curves taken from the ventricle direct show comparatively little trace of the oscillations while the auricle is fibrillating (Fig. 31 *III*).

There is therefore no question but that, in experimental auricular fibrillation, oscillations in the electric curve are produced which may be definitely located as arising in the auricle ; there is equally no doubt that the oscillations are produced as a result of the fibrillation, for they are contemporaneous with it and with no other mechanism.

It is only at the time when this communication is arriving at completion, that the preliminary notice of Rothberger and Winterberg's researches has come to my notice. The curves which they give as illustrating auricular fibrillation in limb leads are of the same nature as those portrayed by the accompanying figures.

The electrocardiographic curves compared in more detail.

One of the outstanding features in the electric curves of both the clinical condition and experimental fibrillation is the presence of oscillations. These oscillations vary considerably in form, in rate and in extent from case to case, and from experiment to experiment. But when the material for selection is abundant it is often possible to choose from the comparates examples of curves which are alike pictorially. Collectively the curves fall into line rather in virtue of qualities which they bear in common.

Rothberger and Winterberg, making independent observations, have recently drawn attention to the oscillations in auricular fibrillation, and have noticed the resemblance of the oscillations found to those seen in cases of irregularity of the heart.

For purposes of pictorial comparison two curves have been selected, and are shown in Figs. 22 and 23. The first is an experimental curve, and the second is from a case of mitral stenosis with completely irregular pulse. The similarity is striking. In instances where the ventricular beat is rapid (Fig. 15), and where on this account the oscillations are obscured, the nature of the rhythm is identified not so much by searching for oscillations as by noticing the deformity of those portions of the curve which lie between the

adjacent peaks R R . The pieces of curve referred to show no resemblance to each other. A similar example but an experimental one is shown in Fig. 17 (the last half of the curve). The first half of this curve represents the escape following vagal stimulation. The oscillations which are so completely unmasked by the prolongation of diastole escape attention in the succeeding cycles. In the clinical curves the peak R is exaggerated, as compared to the normal. The same remark applies to most experimental curves (the comparison may be made in Figs. 20 and 21 and in Fig. 29). The difference is also present in curves taken direct from the ventricle (Fig. 31 *III* and *IV*).

Again, there is the fact that in experimental as well as in clinical curves the general character of the ventricular complex is unaltered. In the dog this can be readily demonstrated by leading from any two points of the ventricular surface. The same type of curve is yielded whether the auricle is fibrillating or in co-ordinate contraction. Fig. 31 *III* and *IV* may be compared. The former, taken while the auricle is fibrillating, shows a faster heart beat to the right and the last phases of the shorter cycles are curtailed. To the left of the same figure, the full complex is shown while the heart is escaping from the inhibitory slowing. The ventricular complexes are of the same form as those exhibited while the sequence is normal, and the heart regular (Fig. 31 *IV*). The same fact may be shown when the oscillations lack prominence. Leads from the upper and lower part of the chest of the same animal gave similar and normal ventricular curves with one or other mechanism present (Fig. 31 *V* and *VI*). The two illustrative curves show the same events, the passage from fibrillation to normal sequence, but in *VI* the galvanometer was arranged to give an excursion approximately three times as great as in *V*.

The electrocardiographic curves, experimental and clinical, are alike in every other respect. The irregular distribution of the ventricular peaks R, the direction of these peaks (direction of base negativity) and the submerged variation T, are features held in common. There is a further characteristic, which deserves more attention. It is common to both clinical and experimental curves. There is no fixed relationship between the heights of the peaks R, and either the pauses which precede them or the height of corresponding carotid beats. The absence of both relationships is shown in Figs 15 and 17. The same disproportions are seen in the condition known as heart alternation*, and it is not improbable that a common factor aids in its generation under the two sets of circumstances. But though I believe a phenomenon of this sort plays some part, yet in certain of the records it is obviously a minor factor. It will be clear that when the oscillations are extensive in amplitude that much will depend upon the relative positions of peak R and summit of auricular oscillation. The individual oscillation may be regarded as Λ shaped ; if a peak R falls where the depression between

* Winterberg[67] has reported some observations from which he concludes that fibrillation leaves behind it, on terminating, a state of altered contractility.

two adjacent oscillations occurs, that peak will be relatively short ; if it falls on the summit its height will be enhanced ; while falling on the side of the Λ a midway position may be expected. Evidence for this view was advanced in the discussion of Fig. 29, and a careful examination of Figs. 22 and 23 substantiates it. The oscillations in these two figures are not spaced with absolute regularity, but it is possible to reconstruct those oscillations which are distorted by R variations coinciding with them. It is found that where a peak R falls at a point at which the summit of an oscillation is to be anticipated the peak is tall in comparison to one which falls where a depression is expected. On the other hand there are many curves in which this explanation is insufficient. Fig. 31 *III*, is an experimental curve taken by means of direct leads from the right ventricle during fibrillation. Oscillations are entirely absent, yet there is the absence of relationship between the amplitude of preliminary variations and the length of preceding pause.

Reviewing the electrical phenomena as a whole it becomes manifest that the clinical and experimental curves resemble each other in every respect. The close pictorial resemblance in many instances, and the absolute identity of all the essential characteristics taken alone or in relation to corresponding events, are in themselves sufficient to carry conviction of a similar mechanism in clinical and experimental instance.

The radial curves compared.

In 1906, Cushny and Edmunds[3 & 4] investigated a case of paroxysmal irregularity of the heart. Unfortunately no venous curves were taken, but from the radial tracings it is in the highest degree probable that the irregular heart action with which they were dealing was of the nature here considered. These authors drew attention in their paper to the close similarity of the radial curves taken from their patient and arterial curves obtained from dogs in which the auricle was in a state of fibrillation. They remarked in particular upon the absence of relationship between the height reached by the arterial beats and the pauses preceding them.

In so far as the experimental arterial curves are concerned. I have little to add to the description given by Cushny and Edmunds. The irregularity of the arterial pulse in auricular fibrillation is absolute and has the same qualities as those presented by the curves in complete irregularity of the heart in man. The rate of the ventricles is increased. Some examples of the Hürthle manometer curves taken from the carotid of a single animal are given in Fig. 6. A short strip of normal curve, the only piece in the figure, is shown in the third line. At the point where the arrow is placed, the auricle, which had spontaneously ceased to fibrillate, was faradised once more and the fibrillation was re-induced. The curves were taken with the chest wall intact and may be compared with the numerous radial tracings given in this paper (Fig. 1, etc.).

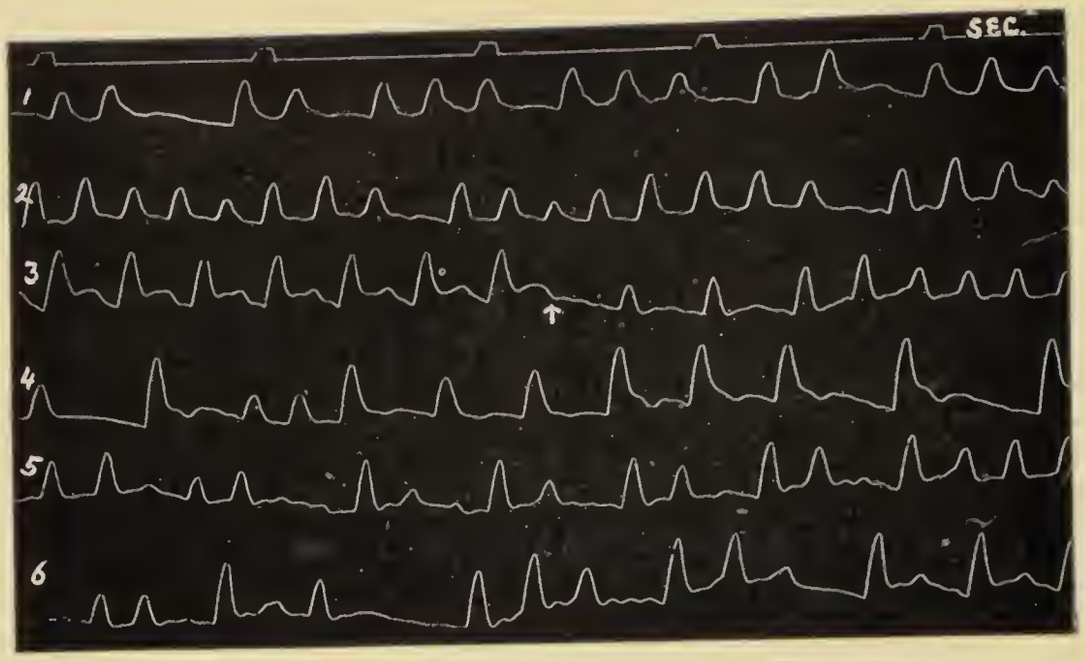

Fig. 6 (1-6). × ⅔ linear. The time marking is in seconds. Hürthle manometer curves from the carotid of a dog in which the auricle was fibrillating. Chest wall intact. A small portion of normal curve is shown in line 3. At the point where the arrow is placed the auricle, which had resumed its normal rhythm, was faradised.

The venous curves and their comparison.

The venous curves associated with complete irregularity of the heart have been discussed at length in the preceding pages.

The venous curves in experimental auricular fibrillation were obtained by exactly similar means*. The ink polygraph was employed, and large and well-fed dogs were chosen for the purpose. The femoral curves were secured by exposing the artery in the thigh and by stitching the receiving apparatus in place over it. The jugular tracings were obtained by fastening the receiver to the shaven neck; the application of vaseline ensured complete closure of the transmitting system. Curves have been taken with the chest open and closed. They present no essential difference; those which illustrate this section are examples from animals in which the chest wall had been restored.

In Fig. 7 *a* and *b* strips of curve from a single animal are represented. In the upper curve the normal rhythm is interrupted by a short induced paroxysm of irregularity due to fibrillation of the auricle. The venous tracing while the rhythm is regular, before and after the paroxysm, consists of clearly inscribed *a*, *c* and *v* waves. During the paroxysmal period each of the irregular beats (several fail to impress the femoral curve) is accompanied by two prominent waves in the veins; the waves are separated from each other, and the second wave is succeeded, by a well-marked depression.

* Hering[20] states in a recent note, that he has seen venous curves, and that they are of ventricular form.

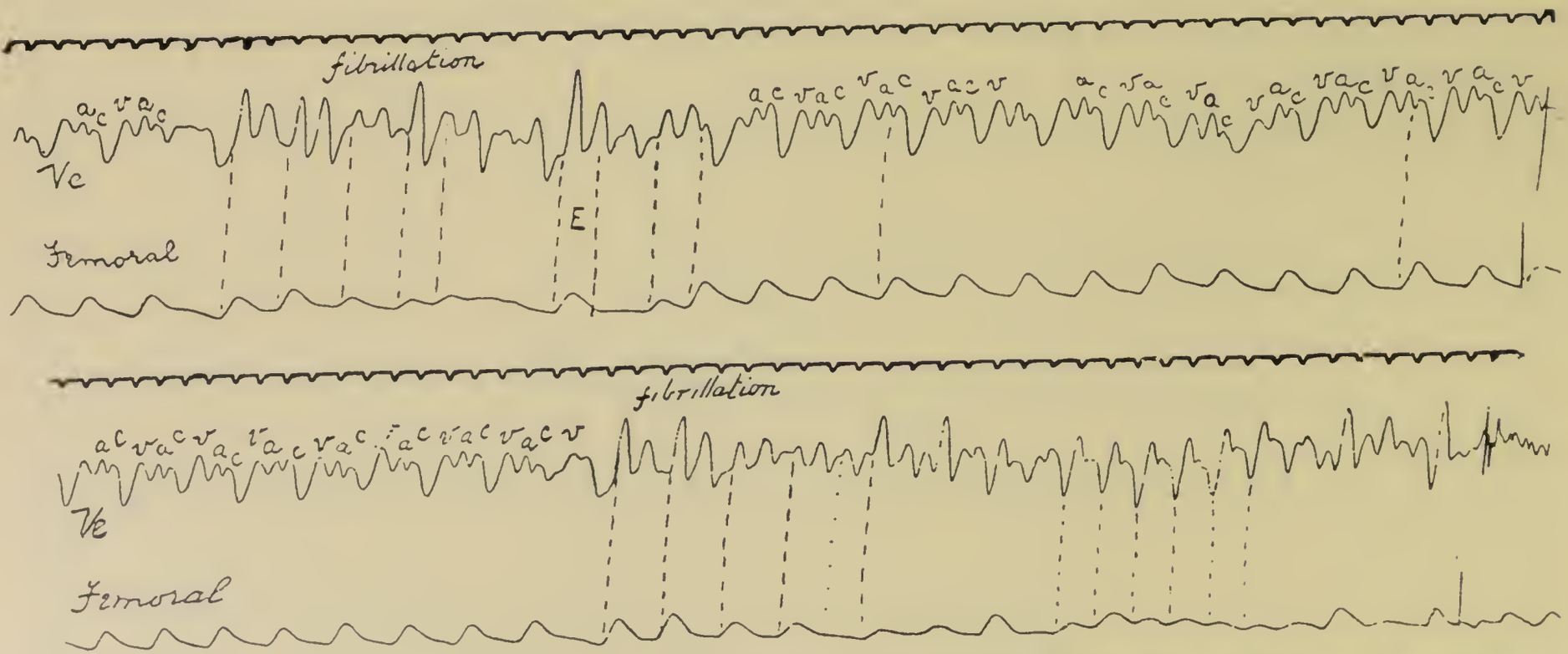

Fig. 7 *a* and *b*. Polygraph curves taken from a dog with the chest wall intact. Venous and femoral curves are portrayed. Each curve shows the normal rhythm, associated, with *a*, *c* and *v* waves, interrupted by a short paroxysm of irregular tachycardia, as a result of faradisation of the auricle. During the paroxysm the venous pulse is ventricular in form.

The main waves fall in the systolic interval, E, but where the pause of diastole is long a stasis wave is also apparent. The lower tracing of this figure shows the onset of a similar paroxysm, and the venous curve at its commencement is of the same type. Towards the termination of the curve there is a succession of more rapid beats and the corresponding venous curves incline to the plateau form ; the systolic depression of the slower beats is wanting. At no point in either tracing are true presystolic waves discoverable while the auricle is fibrillating. The venous records are of precisely the same type as those which characterise the clinical condition. The figures may be compared most profitably with Fig. 2 B and A, and with Fig. 4 B (experimental venous curves, abnormal and normal, are also shown in Figs. 20 and 21). The variation in form with the enhanced rate of ventricular beats has its clinical parallel. Further, in the experience of these experiments, variations have been met with which seem to indicate that the plateau type of venous curve may result from cardiac dilatation, but this feature has received no special investigation. The plateau form is more commonly found towards the end of an experiment. The examples given in Fig. 8 belong to the later stages of an experiment.

Fig. 8 is published to illustrate the effects of vagal stimulation. Irritation of the vagus, while the auricle is in delirium, produces very definite effects. By inspection of the auricle it is determined that the muscular activity is more finely subdivided and more flickering during the time of inhibition. The auricle is more ballooned. At times the fibrillation ceases altogether (Kronecker and Spallitta[28], Phillips[18 & 19]). In my experience the cessation of the delirium, and the return of the normal sequence under vagal stimulation is almost invariable, provided that the fibrillation has been of short duration. But the result is not absolutely constant, and it is less frequent when the inco-ordinate movement has been present for many minutes. The increased distension is invariable. The ventricular rhythm is always markedly retarded (K. and S.), and the rate of beat is slow for some while after the cessation of the stimulation. As the heart quickens again it may do so in response to a fibrillating or to a normally contracting auricle.* When an auricle has been fibrillating uninterruptedly for some while, and the vagus has been stimulated and slow ventricular action has resulted, I have noticed, by no means infrequently, that after the cessation of stimulation and with the re-establishment of rapid response to fibrillation, the latter having continued for a brief period only, gives place to normal or co-ordinate contraction. The retardation of the ventricular beat, while the auricle is still fibrillating is probably due, as Cushny and Edmunds suggest, to a hindrance to the passage of the auricular impulses across the auriculo-ventricular junction. Both the curves which are given in the accompanying figure (Fig. 8) show a return to the normal rhythm

* According as the inhibitory influences have or have not re-established the predominance of the normal pace-maker.

after a period of ventricular slowing. But in each instance a greater or lesser grade of A-V heart-block is demonstrated by the venous curves, after the restoration of the usual sequence. In the upper tracing the *a-c* interval is wider with the first few beats and gradually diminishes up to the point where the stops are seen. The heart-block is of a higher grade in the second tracing. At the escape of the auricle from its fibrillary state it meets with no ventricular response, and during the rest of the figure a 2 : 1 relationship of auricular and ventricular systoles is maintained. The definite sign of obstruction to the passage of auricular impulses and the halved ventricular rate which results in the later stretches of the curve, strongly favour the view that during the fibrillary period the slowing has been brought about in a similar manner, namely by a reduction of the number of impulses conveyed from the upper chamber. Subsequently, the clinical import of these remarks will be more manifest.

The duration of the fibrillation is clearly depicted in the figure. As soon as the pulse rate falls and the diastolic pauses reach an appreciable length, fine oscillations following each other at a rate of approximately 700 per minute make their appearance in the venous curves. They are produced by the fibrillary movements of the auricular wall and vanish at the return of the co-ordinate contraction of its musculature (thus, in the upper tracing the fibrillation ends at the spot indicated by the arrow)*.

The appearance of these rapid and small undulations of pressure in the veins during experimental fibrillation finds its parallel in the clinical case. The waves are not found in all cases, experimental or clinical : a slow ventricular action is essential (cp. Fig. 4 A, and the discussion relating to it). While their dependence upon fibrillation in the experimental case cannot be gainsaid, a similar origin is not demonstrated with equal facility in the clinical instance. At the same time an additional observation is helpful. In man the rate of the venous waves is approximately the same as the rate of the electric oscillations. Simultaneous electric and venous curves are shown in Fig. 19. The oscillations are clear in both lines, and the rate is approximately 450 in each. The rate as portrayed by this figure may be compared with that found in Fig. 4 A, a curve obtained from the same patient on the same day. A comparison between the rates of the experimental oscillations and the experimental venous undulations in the figures already described is not practicable, for the vagal stimulation alters the character of the fibrillation. To the naked eye and in myocardiograph curves the movement appears finer and faster during inhibition. The electric oscillations are also more rapid under these circumstances (see Fig. 17, in which the rate is approximately 800 per minute). In many instances the rapidity is even greater. I have tried on several occasions

* In the lower tracing, the fibrillation ends in what appears to be a full auricular contraction. I have seen the same in electric curves ; it has probably taken place in Fig. 31 *II* ; such beats usually yield anomalous electric curves, and are not the outcome of sinus impulses.

Fig. 8 *a* and *b*. Polygraph curves from a dog. Chest wall intact. Showing the venous and femoral curves while the auricle is fibrillating. During each record the vagus was stimulated. Fibrillation waves in the veins are unmasked and eventually the fibrillation ceases. With the return to the normal rhythm a certain degree of heart-block is present.

to institute a less precarious comparison by severing the auriculo-ventricular bundle, in the hope of obtaining simultaneous venous and electric curves while the ventricular beat is slow and the oscillations prominent, but so far the experiments have lacked success.

It seems plausible to attribute the finer state of fibrillation under vagal stimulation to the phenomenon assigned as the cause of the slowing of the ventricle, namely, to heart-block, at the A-V junction in the one case, and in the walls of the auricle in the other. A conclusion which seems warranted and which is more relevant to the main thesis of this communication is that the venous and electric oscillations, be they clinical or experimental, are the result of one and the same mechanism.

Reviewing the observations upon the venous pulse in complete irregularity of the heart and in experimental auricular fibrillation we may state that the records are alike in every respect.

A summary of the comparison instituted between the records obtained in complete irregularity of the heart in man, and in experimental auricular fibrillation.

In an earlier section it was stated that the proposition advanced, namely, that the two conditions, complete heart irregularity in man and auricular fibrillation in the dog are of one and the same nature, must stand or fall by a careful comparison of the two conditions. This comparison has been completed in the intervening sections. It has been ascertained that the clinical and experimental conditions resemble each other in every respect in which they have been investigated. The observations are summarised in the following table, which consists of a systematic list of the features presented in common.

The radial curves.

1. *Rate increased as compared to the normal.*
2. *Presence of absolute irregularity.*
3. *Absence of fixed relationship between the strength of a beat and the preceding pause.*
4. *The presence of many "dropped" beats, and of beats of all shapes and sizes.*

The venous curves.

1. *The presence of the ventricular form of venous pulse (the absence of "a" waves).*
2. *The appearance of definite variations in the form of the individual venous pulse curves.*
3. *The presence during diastole of rapid undulations of venous pressure when the heart beat is slow.*

The electrocardiograms.

1. *The occurrence of a base negative variation R at the commencement of ventricular systole.*
2. *The presence of a variation T which is deformed in leads from the extremities, but which is clear cut and of normal form in leads from the heart itself.*
3. *The absence of an auricular systolic variation P.*
4. *The occurrence of continuous oscillations, generated in, or in the vicinity of, the auricle. The rapidity of the oscillations. Their disappearance when the normal rhythm is resumed.*
5. *The absence of fixed relationship between the height of the peak R and the length of the preceding heart pause. The absence of relationship between the height of R and the height of corresponding arterial curves.*
6. *The increase in the size of R as compared to the similar peaks of the normal curve.*

Auricular fibrillation as a clinical entity.

The clinical and experimental comparison employed as a deliberate test of the proposition, originally based upon purely clinical facts, is invaluable. Without it our attitude towards a large group of clinical cases remains in the hypothetical stage. With the comparison hesitancy gives place to a feeling of security, and we may approach our patients with a true appreciation of the affection which we so frequently encounter. The meaning of many otherwise obscure manifestations becomes clear, and we are in a position to pursue the path of investigation towards the hoped for cure or prevention with the sense of a firm footing.

The possibility of the occurrence of auricular fibrillation as a clinical phenomenon has been recognised by several writers. As we have seen, it was suggested by Cushny and Edmunds.* Their suggestion, based as it was upon the comparison of the arterial curves in a single clinical case with those obtained experimentally, has not developed up to the present time; the hypothesis was a tentative one. In the discussion at the end of their paper they state: "Of course we cannot claim to have shown definitely any connection between this type of irregularity in the dog's heart and that in the case described. At the same time there exist similarities between them, and the sudden onset of the irregularity in each case suggests a common cause;" Wenckebach[65], writing a year later, briefly discussed the possibility.

In 1908, Hirschfelder[24] published some notes on auricular fibrillation, and concluded that certain cases of paroxysmal tachycardia were the result

* The first suggestion came from Cushny (*Journ. Exper. Med.*, 1899, IV, 340).

of increased auricular irritability. The auricular tachycardias to which he refers are of an essentially different nature to the disturbance of heart rhythm which we are discussing, and there is no reason to suppose, but on the other hand every reason to deny, that they are the result of auricular fibrillation in the true sense of the term. But when the auricle is faradised with a weak current, a preliminary quickening of rhythm takes place, and as the stimulus is increased and spreads fibrillation may ensue. In this limited sense a connection may be traced between regular auricular tachycardias and auricular fibrillation, and I have met with a single instance in which the one condition (regular auricular tachycardia) passed directly into the other (complete heart irregularity or auricular fibrillation). (This instance is subsequently described ; Fig. 9 and explanatory remarks*.)

It may be said, therefore, that although auricular fibrillation has been regarded by certain isolated observers as a possible phenomenon in clinical pathology, its association with anything beyond rare cases of paroxysmal tachycardia has not been seriously attempted until the last few months. The introduction of the string galvanometer as an aid to diagnosis has facilitated a much wider conclusion. While these investigations were in progress this conclusion has been partially and independently arrived at by Rothberger and Winterberg[36], working in Vienna. In their preliminary communication they conclude that certain cases of *pulsus irregularis perpetuus* are due to fibrillation. A large clinical experience of such cases exempts me from the necessity of restricting my conclusions, and permits me to assert that all such cases belong to a single group, and that the mechanism is the same in all.

Where, in conjunction with an absolutely irregular pulse, the ventricular form of venous pulse and the characteristic electrocardiogram are present, the evidence is complete. But, as in a large number of patients no case, exhibiting complete irregularity and the ventricular form of venous pulse, has been found, which does not show likewise the typical electric records, the electrocardiograms cannot be held as essential to the diagnosis. Moreover, as no case of complete irregularity has been met with, or has been recorded by any other writer, in which the venous pulse when given may not be interpreted as of the ventricular form, and as the cases in which these venous and arterial signs are present together have been published in abundance, the conclusion may be carried a stage further. There is no hesitation in stating that in the vast majority of cases of complete irregularity of the heart, auricular fibrillation is responsible for the disturbance of ventricular rhythm. It is sufficient if a single strip of curve is obtained from the radial artery, and if no two beats on the strip are of the same length (and given, of course, that the heart rate is not manifesting a gradual acceleration or retardation while such a strip of curve is recorded) then the statement that auricular fibrillation is present is justified in all but extremely

* Hewlett[22] has reported a case which was very possibly of the same nature.

exceptional cases*. It may be affirmed that auricular fibrillation accounts for the largest class of persistent heart irregularities ; a class which, broadly speaking, composes 50 per cent. of all such irregularities. And in dealing with cases of mitral stenosis admitted to hospital with failing compensation, it may be said that the presence of auricular fibrillation is the rule.

In discussing the question of the possibility of auricular fibrillation, I have been frequently met by an argument of a purely *a priori* nature ; and, judging from the note by Rothberger and Winterberg, these observers appear to have had a similar experience. It is said that it is inconceivable that the heart should remain in this state for long periods. It should be known that Janowski[25] recorded a case of complete irregularity of 5½ years' duration, and Mackenzie has watched similar patients for even double this time. In reply Rothberger and Winterberg instance the persistence of tremor in the tongue following nerve section, and I may draw attention to the well-recognised and chronic fibrillary twitching of the voluntary musculature in many nervous maladies†. Fibrillation of the ventricle it is true is accompanied by almost instant dissolution, but fibrillation in this chamber brings the circulation to a prompt standstill. The auricles are not indispensable to the circuit of blood in the body, but may be regarded rather as temporary reservoirs which accommodate the blood flowing to the heart while the ventricle is in systole. The large veins are perfectly capable of subserving this function, and the resulting general disturbance of the circulation is but slight. We have perfectly definite evidence that the normal auricular contraction is in abeyance in a large number of patients in whom the peripheral circulation is unhindered, and auricular fibrillation may last for many hours in a dog and yet the blood-flow in the body is fair throughout. Therefore, on the ground that auricular fibrillation is incompatible with a continued circulation, this argument may be entirely dismissed. If it is implied that the hindrance would occur, not in the auricle, but in the ventricle in virtue of the high grade of disorder of the rhythm, it may be replied that such an argument is but a denial of an ascertained fact. The secret of the lack of disturbance as

* The only exception, I think, is the rare case of gross sinus arrhythmia, independent of respiration.

† I had hoped to have obtained direct evidence upon this aspect of the question, but no opportunity has presented itself. I am convinced that a similar affection is found in the lower animals, and have seen one example of it. Examination of the heart *in vivo* was not practicable ; sooner or later, it is hoped, observations in this direction will be forthcoming.

Since these pages were written a horse has been obtained, through the kindness of Professor Woodruff of the Royal Veterinary College, in which electrocardiographic curves demonstrated complete irregularity and an absence of P variations. The horse was killed and the chest quickly opened. Unfortunately the right ventricle was damaged during this operation. Inserting the hand into the chest the ventricle could be felt to beat rapidly and irregularly. No movement could be felt in the right auricle. The ventricle was grasped and drawn through the wound, but fibrillation ensued. The auricle was then seen in a state of fibrillation, and it continued to fibrillate until all movement ceased. The necessary repetition of this observation will be undertaken so soon as opportunity presents itself.

a result of the condition *per se* seems to me to be found in the direction of propagation of the wave of contraction in the ventricular musculature. The electric records testify that the contraction route taken is the normal route. Strong and weak contractions may be mixed together in profusion, but all the beats will tend to be effective in expelling the ventricular contents, and the majority of the beats are effective.

Even if the *a priori* arguments could not be met, the evidence for the proposition is so positive that we should be justified in setting them aside. A case is recorded in this communication (*CASE 12*), in which incontrovertible evidence (arterial, venous and electric) is at hand to prove the presence of the fibrillation; and, be it noted, this case has been under observation, and has presented the same mechanism, as shown by arterial and venous curves, for a space of five years.

Auricular fibrillation and heart-block; the action of digitalis.

It is by no means uncommon to meet with clinical cases of complete irregularity in which the ventricular action is not excessive, and instances are not infrequent in which the rate is actually reduced. Moreover it is well known that in complete irregularity of the heart retardation of the ventricular rate constitutes one of the most characteristic actions of drugs of the digitalis group. In consideration of the fact that the sinus rhythm is in abeyance the cause of the slowing must be sought in a part of the musculature other than that at which the pace-maker is situate.

In discussing the experimental venous curves, the question of ventricular slowing under vagal stimulation received attention, and facts were brought forward in favour of the contention that the retardation is the result of the blocking of fibrillation impulses at the A-V junction.

Now we have proofs that digitalis acts upon the vagus and that the vagus has a powerful influence upon A-V conduction. Further, we have very suggestive evidence that digitalis may act *directly* upon the junctional tissues between auricle and ventricle. The facts upon which these conclusions are based have been recently examined in a communication to the *Quarterly Journal of Medicine*[29]. Their relevance to the phenomenon of ventricular slowing in clinical fibrillation should be obvious; it may be surmised that digitalis slowing is produced either by a direct action of the drug, or by an indirect action through the vagus, upon the junctional tissues. We may examine the remaining evidence which supports this view.

We know that in rheumatic heart affection, and in mitral stenosis in particular, it is common to find signs of imperfection of conduction of the contraction wave from auricle to ventricle (see *CASE 11* and *12*), and we are also aware that in such cases it is the rule that the exhibition of digitalis or allied drugs enhances the grade of heart-block (Mackenzie[36] and others). We know further, as a result of Mackenzie's work, that of cases of auricular

fibrillation ("nodal rhythm"), those which are of rheumatic origin or those in which mitral stenosis is present are most susceptible to digitalis. Therefore the suggestion which is put forward is that the slowing of the heart, when auricular fibrillation is present and digitalis is given, is due to an increase of a previously existing defect* in the conduction to the ventricle of those impulses which are built up rapidly and irregularly in the auricle. We require facts from two sources in confirmation of this view. First, experimental data are necessary, and at present these are not forthcoming. Secondly, we require more extensive observations which will show rather than suggest that, in those cases of fibrillation in which digitalis produces retardation, conduction was impaired before the onset of the new rhythm. Or, more strictly perhaps, it will be necessary to show that conduction was primarily impaired in a much larger percentage of cases which react than of cases which fail to react, for it is possible that fibrillation may be the forerunner of conductivity changes in some instances. These statistics will be slow in coming, but already there are indications in harmony with the proposition. It is the rheumatic case which usually reacts, and it is the rheumatic case which usually shows altered conduction. Again, taking the cases which are recorded in this paper, three of the patients observed before the onset of fibrillation, or during the intervals between paroxysm, have been thoroughly tested from this point of view. Two of the cases (*CASES 2* and *10*), in which the *a-c* or P-R intervals were of normal length during the normal sequence, failed to react†, while the third, a case (*CASE 11*) in which conductivity was definitely impaired, reacted in the most typical manner.

We have seen that two allied phenomena, increased heart-block when the A-V rhythm is present and retardation of the ventricular rate in auricular fibrillation, may occur as a result of digitalis. A similar parallel may be drawn between heart-block arising in the absence of drug administration and the spontaneous slowing which not infrequently occurs in complete irregularity. Moreover cases are on record in which the irregular rhythm is slow from the outset. A number of patients with slow and irregular heart action have been collected and described recently by Mackenzie[40] under the term "nodal bradycardia," and they form an extremely interesting group. The examination of one or more such cases with a view to ascertaining the nature of the auricular action became crucial. As a result it has been found that auricular fibrillation is present in these instances also; the two cases examined may be cited. The details of these patients are most instructive, but they can be alluded to but briefly. Both cases belong to the series reported by Dr. Mackenzie, and I am indebted to him for the opportunities of re-examining them. Fuller clinical reports will be found in his communication to *this Journal* (p. 23).

* The defect may be a potential one in certain instances.

† Digitalis was administered until sickness resulted.

CASE 12.—The following is an abstract of the original report (*Heart*. Vol. I, p. 25). W. H., a subject of rheumatic fever, was known to have had impairment of conduction in greater or lesser degree for 12 years. He then suddenly developed slow and irregular action of the heart and demonstrated the ventricular form of venous pulse. In this state he continued a week, at the end of which time the pulse was again regular and the *a*-*c* interval was 0·4 sec. in length*. Seven months later the same slow and irregular action of the heart was resumed and it has persisted until the present time, a period of five years.

A tracing from this patient is given in Fig. 4 A. On the day upon which he came for re-examination the pulse rate was 49-68. The pulse was absolutely irregular, the venous curve being of the ventricular form. There were fibrillation waves in the curves. In Fig. 19 simultaneous electrocardiographic and venous curves are shown. The electrocardiographic record is characteristic and the usual oscillations are present.

There can be no question but that the case is one of auricular fibrillation. The long-continued history of heart-block previous to the onset of the irregularity and its presence during a brief interlude strongly supports the view that the slow ventricular rhythm resulted from the known inefficiency of the junctional tissues which transmit the irregular impulses formed in the auricle.

CASE 13.—M. M., age 45 (extract from *Heart*, Vol. I, p. 33).—"Permanent nodal rhythm, bradycardia associated with mitral stenosis. Occasional attacks of syncope and convulsions."

Recently the case has been fully re-investigated, and a detailed report of it will be found in the contemporary number of the *Quarterly Journal of Medicine*. The main facts in regard to the patient are as follows :—Apart from the epileptic seizures the pulse rate is almost constantly at or about 30 per minute. This is the rate generally assumed by the ventricle when it is entirely dissociated from the auricle. As in complete heart-block the pulse is *regular*. The patient has syncopal and epileptic attacks in every way similar to those met with in the subjects of complete heart-block. Extrasystoles, when they occur, are followed by pauses equal to the spaces between adjacent beats of the usual slow rhythm. The patient has had syphilis.

Briefly, the patient presents a picture identical with that of complete heart-block, as usually recognised, in every respect but one. There are no co-ordinate auricular contractions. The venous pulse is of the ventricular form and electrocardiograms from the extremities and chest wall (Fig. 18 and explanation) afford clear evidence that the auricles are fibrillating.

The conclusion is unavoidable that the case is one of complete heart-block (the result of syphilis) and auricular fibrillation. The regularity of the pulse is a natural consequence of the inability of the auricular impulses to reach the ventricle. Frederieq[9] has shown experimentally that section of the bundle, while the auricle is fibrillating, cuts off all auricular impulses.

Dr. Gibson, of Edinburgh, has drawn my attention to a case, of considerable interest in this connection, which he reported in 1906. The tracings

* The normal *a*-*c* interval is 0·2 sec. or slightly less.

obtained were those of incomplete heart-block (the auricular rate was 168 and the ventricular 42). On one occasion there was a temporary disappearance of the auricular venous waves, and *while the pulse became completely irregular, though still phenomenally slow, faint and rapid oscillations appeared in the venous curve.* (The rate of the oscillations was approximately 375 per minute, as shown in the published curve[11]). A post-mortem revealed an increase in the fibrous tissue of the bundle with wide separation of the fibres constituting it*.

I think there can be no reasonable doubt that here also auricular fibrillation and heart-block were present in combination, and the most important evidence for it is the *type of irregularity* which the pulse assumed when the regular auricular waves vanished.

Reviewing the three preceding cases we may conclude that auricular fibrillation occurs clinically in association with heart-block, either partial or complete. The case for a similar mechanism under digitalis is strengthened. And I would go further and put forward the general hypothesis that when auricular fibrillation is present and a slow ventricular action is found, whatever its cause, a certain degree of temporary or permanent impairment of the functions of the junctional tissues may be assumed.

A tabulated statement of the suggested or proved relationships between heart-block and auricular fibrillation may be given in summing up the conclusions of this section.

A-V RHYTHM.		*AURICULAR FIBRILLATION.*	
Heart-block.	*Digitalis H.B.*	*Heart-block.*	*Digitalis H.B.*
Lengthened a-c interval	*lengthened a-c interval*	*spontaneous slowing of irregular tachycardias*	*ordinary digitalis slowing*
2 : 1 ; 3 : 1, etc., heart block	*2 : 1 ; 3 : 1, etc., heart-block (Mackenzie[36])*	*spontaneous slowing of marked grade, and cases with onset of very slow and irregular rhythm (CASE 12 and Gibson's patient)*	*marked slowing on digitalis (Mackenzie[36])*
Complete heart-block	*complete heart-block†*	*auricular fib. and complete heart-block (CASE 13)*	*digitalis slowing with the production of a regular pulse of 30-40 per minute ("Heart," Vol. 1, p. 39)*

* I may add that Dr. Gibson writes me that it occurred to him at the time that the auricles might have passed into fibrillation. A somewhat similar case has been reported by Herxheimer and Kohl (*Deutsch. Archiv f. Klin. Med.*, 1910, XCVIII, 330), but the facts, I believe, have been misconstrued.

† Tracings from a case of temporary complete heart-block the result of the administration of strophanthus were shown to me lately by Dr. Emanuel of Birmingham.

The cause of the onset and duration of auricular fibrillation.

The primary object of this paper is to establish the fact that auricular fibrillation is a frequent clinical affection. Once established our duty lies in the direction of seeking its cause. In this respect I am unable to offer more than incomplete evidence, and the views expressed are at present almost purely hypothetical.

It will be generally allowed that fibrillation consists in the elaboration of numerous and fresh impulses in the auricular walls. It may be regarded as a state in which stimuli are generated, at many separate and uncertain points and the inco-ordination of the contracting fibres may be held to result from the impact of contraction waves and the production of localised areas of block. The condition may be compared in many respects to the confusion of uncontrolled traffic in a crowded thoroughfare. In seeking for an explanation of the origin of auricular fibrillation we naturally turn to our knowledge of the causes which originate new impulses. That, at the present time, is the most important problem relating to cardiac irregularities.

Now it has been shown that an increase of intracardiac pressure is succeeded by the appearance of ectopic impulses. Hering[14] employed this method in his studies of extrasystoles. Auricular fibrillation is specially prominent in cases of mitral stenosis ; the high intra-auricular pressure in this disease might be considered sufficient to account for its occurrence. I have observed the onset of auricular fibrillation in experiment on several occasions when, by pressing on the abdomen, the venous inflow was suddenly increased. But the difficulty of accepting the view as a general hypothesis is obvious, for auricular fibrillation occurs in many cases in which a raised auricular tension may not be assumed.

A purely mechanical doctrine is untenable. The cause must be sought in a more detailed study of the morbid anatomy, and attention should centre in the auricular walls. So far we have little information. Several cases of gross irregularity of the heart were investigated by Radasewsky[30] some years ago, and he found widespread fibrosis of the heart. He remarked that the damage was most extensive in the auricles. Mackenzie[39] has given post-mortem notes of eight cases of " nodal rhythm " : in three the auricle showed fibrosis, in two reports it was not mentioned, in one not examined, in one the septum was " stretched," in one atrophied. The ventricle, on the other hand, showed fibrosis in seven instances : in one it is not mentioned ; in six of the cases the junctional tissues were fibrosed or otherwise damaged. It is obvious from the accounts that the node of Tawara and the bundle received more attention than the rest of the heart. Schönberg[38] has recently examined five hearts from patients with persistent irregularity : in one case venous and radial curves are available[12], and from his clinical accounts the remainder were probably of the same nature, namely, complete irregularities. He found a chronic inflammatory (lymphocytic) infiltration of the tissues at the junction of superior cava and auricle in all. He asserts that

this was the chief lesion ; but here again it is obvious that attention has been concentrated upon a limited portion of the musculature. Amongst the cases belonging to this investigation one only has come to autopsy. It was a case of streptococcic endocarditis, and a large area of ulceration was found on the septal wall of the left auricle. I have since seen, through the kindness of Major-General F. Smith, a case of irregularity in a horse. The irregularity was complete, as shown by tracings, and a systolic pulsation was apparent in the veins of the neck and reached the angle of the jaw while the animal was standing. Great breathlessness and dropsy were present. The disturbance of the heart's rhythm was of eight months' duration. An examination of the heart showed considerable hypertrophy of the ventricles and auricles ; it was especially marked in the latter. The endocardium of the left ventricle and auricle was opaque and thickened : there were large and scattered patches of sub-pericardial fibrosis in the left auricle, but in no other part of the musculature. (The histology will be reported at a later date.)

It is possible that fibrosis, by interfering with the circulation in restricted areas of the musculature, sets up a state of irritability. The tendency for anæmia of the muscle to bring about new impulse formation is readily demonstrated experimentally. Ligation of a branch of a coronary artery is usually followed by the appearance of extrasystoles[10b]. But it is difficult to suppose a constant state of anæmia in the affected tissues, and an anæmia of long duration. It must also be remembered that extensive fibrosis may occur without irregularity[17].

Some light seems to be thrown upon the question by an examination of the relation of fibrillation to extrasystoles.

There appear to be two types of case of the paroxysmal affection. A type in which the interludes are characterised by a perfect regularity of the heart (*CASE 10*), and a type in which the regular rhythm of the slow period is interrupted by extrasystoles (*CASES 2* and *11*).

In my own limited experience the extrasystoles which interrupt the slow periods, or those which interrupt the normal rhythm prior to the onset of the fibrillation, have been auricular in origin (*CASES 2* and *11*). Their presence in these patients and in certain of those reported by Mackenzie arrests attention. We cannot divorce the problems of the factors influencing the origin of single and multiple ectopic impulses, neither can we separate these questions from that of the origin of fibrillation. The production of a single new impulse, of a series of impulses from a single focus, or of multiple impulses from scattered foci, is a matter of degree. Furthermore, we cannot exculpate these single ectopic beats. They appear to participate in producing the ultimate crisis. In the cases observed they have been very numerous. In the last number of this *Journal* a case was described in which auricular extrasystoles were as numerous as normal beats, and in this patient paroxysms of ectopic beats were seen, which also arose in the auricle. On one occasion while a paroxysm was in progress the auricle passed into fibrillation ; and after its duration for about half a minute the auricular paroxysm reappeared,

A portion of the curve, is reproduced in Fig. 9. To the left is the fibrillation period, and to the right are a few beats of the paroxysm for comparison. The original curve is too faint for publication, but the two sections of curve have been traced by a skilled draughtsman and the original is well portrayed. A stop and two inches of curve have been excised at the point where the vertical line is placed. The change from one mechanism to the other was not recorded.

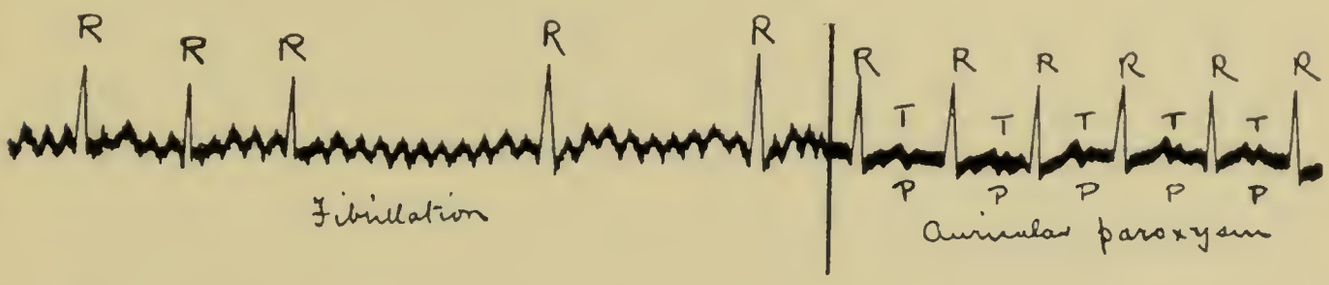

Fig. 9. H. B. (12th Aug. 1909). From a case fully recorded in the last number of this *Journal* (page 262). The original curve has been retraced, and two inches of curve have been excised. To the left the electrocardiograph shows auricular fibrillation. To the right is a paroxysm of regular tachycardia, starting, not at the sinus, but in the auricular walls. This fact is recognised because the auricular variations are absent in their usual form. The peaks R of this paroxysm are not longer than those of the preceding fibrillation period, yet they were always longer than the peaks R of the normal rhythm when the two could be compared. The comparison is utilised as evidence that the peaks R of fibrillation are higher than those of the normal rhythm.

Now in the case of regular auricular paroxysmal tachycardia the actual paroxysms are usually if not always foreshadowed by the occurrence of single or multiple ectopic beats of a different kind. This has been observed invariably in the paroxysms of two cases of auricular tachycardia (*Heart* I, p. 262 and *CASE 15*, with Fig. 26 of this paper). And I have advanced the view that the preliminary beats stand in casual relationship to the paroxysms themselves. If single or multiple beats arising in the auricle may predispose to the production of a paroxysm of regular beats also arising in the auricle, may not these beats also account in part for the onset of an attack of fibrillation? The question arises as to whether a beat arising *de novo* in a chamber of the heart and pursuing an abnormal course in this chamber will enhance the irritability of the musculature in the immediate neighbourhood. Supposing that two or more extrasystoles are started at the same time at different points in the musculature of an irritable auricle, are not those factors present which are calculated to produce a condition of absolute inco-ordination?

The evidence is at present only suggestive: long paroxysms of rapidly recurring beats are apparently provoked by other beats of a similar nature,

but springing from a separate focus (*CASE 15*). The suspicion is awakened that an extrasystole enhances the irritability of the tissues affected, and that their presence in numbers may predispose to if not actually determine the culminating inco-ordination. Furthermore, it is possible that fibrillation, when once produced under suitable circumstances, may itself maintain the increased irritability, and thereby tend to the continuation of the inco-ordinate state. Whether this is so or not should be determinable experimentally, and there are already facts which support an answer in the affirmative.*

Fibrillation is readily produced by faradisation, but after a short period of faradisation the return to the normal rhythm is almost immediate. If the muscle is stimulated periodically and paroxysms of fibrillation result, it is common to find that each successive stimulation gives a more permanent result, that is to say, a result outlasting more and more the actual stimulation, and often a final stimulation will produce a long continued state of inco-ordination. Moreover, the longer paroxysms seem to be less under inhibitory control. Thus it may be held that the fibrillation itself aggravates the irritability of the auricular tissue. It is perhaps a factor of this nature which accounts, partially at all events, for long continued auricular inco-ordination in patients who are affected with it.

Auricular fibrillation and ventricular extrasystoles.

Radasewsky found diffuse areas of fibrosis, not only in the auricle, but also in the ventricle, in cases dying with gross heart irregularity. Mackenzie reported similar changes in "nodal rhythm." Dr. Koch, of Freiburg, tells me that in cases of complete or "chronic" irregularity his experience is similar. We should therefore anticipate that in diffuse fibrosis impulse formation would be frequent in the ventricle. This is actually found to be the case. In the intervals between paroxysmal attacks or before the onset of permanent irregularity, ventricular extrasystoles† have been recorded by Mackenzie. But, as opposed to the possibilities in the auricle there is no reason why co-ordinate ventricular responses to such impulses should not occur when the fibrillation is established. They are not infrequent. An example has been recorded by Hering[18], and in the present series of thirty cases examined electrocardiographically, they were exhibited by seven. They are recognised mainly by the electrical variations to which they give rise, and by the fact that they do not affect the arterial pressure in the same measure as do the ventricular beats which result from auricular impulses.

* It would be of interest to know whether, in cases in which extrasystoles are frequent while the pulse is slow, the periods of irregular tachycardia are of longer or shorter duration than in those patients in whom the slow pulse is uninterrupted in its regularity.

† Analogy would suggest the occurrence of ventricular fibrillation also. Unexpected death in patients with fibrotic hearts is a well recognised fact, and it is not uncommon in patients with auricular fibrillation (*CASE 4*).

A notable example is shown in Fig. 24. The responses to auricle are denoted in the usual way, the first variation is marked R. The remaining beats are ventricular extrasystoles without exception: (the extrasystoles are marked E and the variations are distinguished by letters and figures indicating direction and order, thus *n*1 signifies first base negative variation). In the figure none produce an effect upon arterial pressure. Auricular extrasytoles may be placed out of court* while the auricle is fibrillating, and this is clearly demonstrated by the general character of the remainder of the curve and the occurrence of oscillations *f f*.

Considering the variations presented by the unusual type of beats alone, they correspond in outline to those obtained by stimulation of various areas of the ventricular musculature.†

Such extrasystoles are seen most commonly when the ventricular rate is relatively slow.

Now when digitalis is administered in large doses to patients who are suffering from complete irregularity of the heart, a bigeminal action of the ventricle is often observed when the retardation is marked. The pulse still presents irregularity, but it may happen that the shorter pauses of the bigeminy are of almost or quite constant length. This constancy of length is explained if we assume that the second or weaker beat of the bigeminy is a response to a ventricular impulse and is independent of auricular impulses.‡ An opportunity of putting the matter to the direct or galvanometric test has not occurred.§ The same type of irregular "bigeminy" is met with apart from digitalis administration, and an example of it is shown in Fig. 4 *C*.

Certain conditions differentiated.

Cases in which the ventricular form of venous pulse occurs, but in which either auricular fibrillation is absent or in which the evidence is insufficient to justify us in assuming its presence.

The conclusion has been formed that of cases manifesting a ventricular form of venous pulse those which show complete irregularity of the heart belong to a single group, that of auricular fibrillation. It has also been

* It must be noted that abnormal ventricular variations may occur in beats arising as a result of auricular impulses[23] but the remaining arguments and observations prohibit such a supposition in this instance.

† Two types have been figured by Kraus and Nicolai[27] and have been allocated to right and left ventricle. But I may state as a result of observations as yet unpublished that no two points of stimulation give precisely the same picture, that a practically constant type is given from any single focus of stimulation, and that such type may be held within certain limits as characteristic of the focus or area in which it arises.

‡ The constancy of the short pause will receive the same explanation as that adopted in other cases of accurate coupling arising as a result of ventricular extrasystoles.

§ Electrocardiograms have since been obtained from two patients exhibiting the coupling in question. In each instance the second or smaller beat yielded an anomalous electric complex. The explanation of the coupling adopted in the text is therefore demonstrated as the correct one.

demonstrated that auricular inco-ordination may be present, and yet the pulse regular. But there are at least two types of case in which, while there is no venous sign of auricular contraction and the pulse is regular, auricular inco-ordination is certainly or probably absent. Such being the case, an opinion that auricular fibrillation exists is not justified, where the pulse is regular, in the absence of electrocardiographic records.

The first type is that in which the pulse rate is usually of normal or but slightly increased rate. The patients are not infrequently encountered, and offer signs of dilatation of the right heart with considerable distension of the veins. An example of a polygraph tracing is shown in Fig. 10.

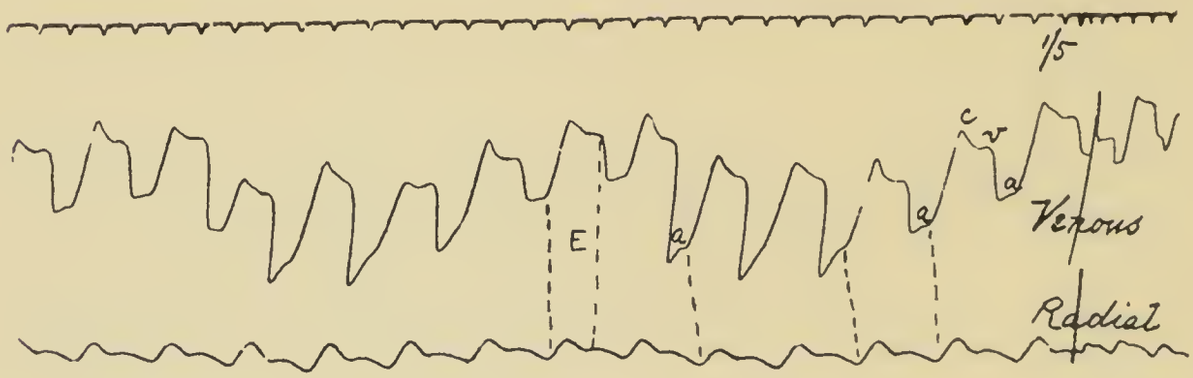

Fig. 10. The ventricular form of venous pulse in a patient with regular action of the heart. Auricular fibrillation was not present (*CASE 14*).

CASE 14.—F. W., a girl aged 12, had suffered from three attacks of rheumatic fever, and there was a clear history of pericarditis. The symptoms consisted of severe shortness of breath, cough and pain in the chest and upper abdomen. On examination the veins were seen to be greatly distended and the tension in them was increased. The pulse was regular and "water hammer" in character, capillary pulsation was present. The liver was enlarged and pulsatile, dropsy of the feet and ascites were found. The right limit of cardiac dulness was 2 inches and the left 6 inches from the mid-sternal line. There was definite post-sternal dulness on a level with the second and third ribs. The lungs failed to cover the heart during deep inspiration and the heart's apex was fixed. An early diastolic murmur was audible over the greater part of the precordium, being maximal at the aortic cartilage. An occasional faint presystolic murmur and a constant systolic murmur were heard at the apex.

Aortic regurgitation, mitral stenosis and pleuro-pericardial adhesions, probably extending to the mediastinum, were diagnosed. The pulse was invariably regular, the jugular curves failed at almost all times to show any trace of presytolic elevation. In the tracing given (Fig. 10) a very faint trace of *a* wave is visible, but it was never more marked than here depicted, and usually could not be obtained. The electrocardiogram demonstrated a clear P variation (Fig. 25). The case is an example of the ventricular form of venous pulse accompanying the normal sequential chamber contraction. (Similar cases have been reported by Hering[16] and Hewlett[21].)

The second type of case is that of a not uncommon form of paroxysmal tachycardia.* I have met with three cases of a similar if not identical nature during the last six months. In one case mitral stenosis was found; in the other two no physical signs were apparent in the heart except during the paroxysms. The paroxysms were associated in the last two cases with slight but progressive dilatation, and the throbbing in the veins of the neck was sufficiently forcible to suggest arterial pulsation. Tracings are given from one of these patients in Fig. 11*a* and *b*. Fig. 11*a* was obtained between two paroxysms; Fig. 11*b* during a paroxysm. In the first curve the venous pulse is of the auricular and in the second of the ventricular form.

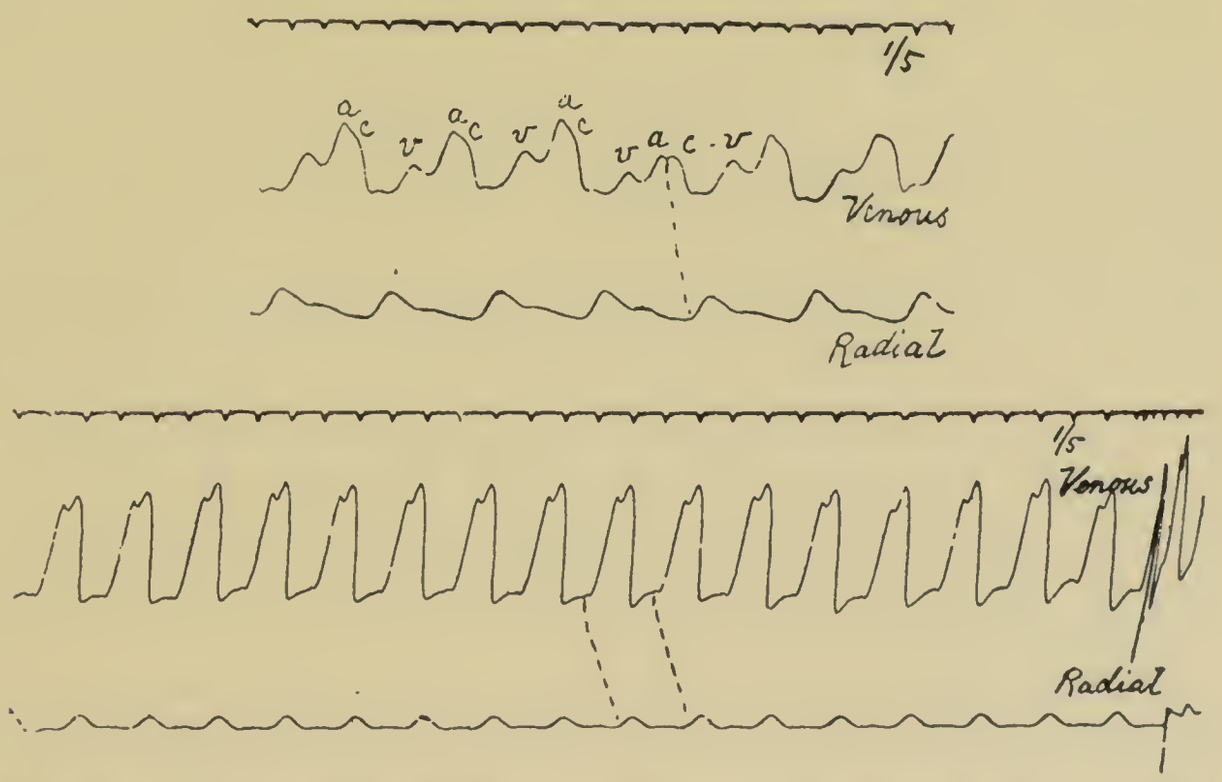

Fig. 11*a* and *b*. Two polygraph tracings from a patient with paroxysmal tachycardia. Tracing *a* is normal. Tracing *b* shows the venous pulse to be of the ventricular form while the paroxysm is in progress. The pulse is regular in both curves.

In both curves the arterial pulse is regular, the respective rates are 92 and 147. In the absence of electrocardiographic curves it is impossible to ascertain the nature of the mechanism. It may be of a kind suggested by Wenckebach[66], for it is possible that with an increased As-Vs interval the auricular contraction falls back on the preceding ventricular systole. In the case of mitral stenosis, previously referred to, this explanation would suffice, for a prolongation of the *a-c* and P-R intervals was present while the pulse

* Certain regular paroxysms accompanied by the ventricular form of venous pulse and described by Mackenzie[39] and Howlett[22] may be included in this group.

was slow. In the patient from whom the figured tracings were taken no impairment of conductivity was ever noticed and the increase of rate was not great. We may perhaps be dealing with paroxysms starting in the ventricle.

The point of importance at present is the fact that interpretations other than auricular fibrillation suggest themselves, and that while the pulse is fast, but regular, the latter may be rationally excluded.

True "nodal rhythm" is a rare affection and gives rise to a clinical picture entirely at variance with that of the disorderly action of the heart hitherto considered.

In the preceding pages it has been demonstrated that the condition which has hitherto passed in this country under the name of "nodal rhythm" is in reality due to an inco-ordinate action of the auricle known to experimentalists as auricular fibrillation. In the discussion based on purely clinical data it was stated that it could be shown that auricle and ventricle are not contracting together. There are many reasons which militate against the acceptance of the view of synchronous contraction of the two chambers in complete irregularity of the heart. These reasons I do not propose to consider, for they will readily suggest themselves as a result of the observations which have been discussed. At the same time I desire to place on record, for purposes of contrast, a single and fully considered instance of what I regard as a case approaching as nearly as possible to what may be termed, in the present state of our knowledge, true nodal rhythm.* It will be shown that a rhythm may be generated in the auricle near to or at its junction with the ventricle, and that the clinical picture to which it gives rise is strikingly at variance with that which is presented by complete irregularity of the heart.

The following case is the only one of its kind which I have met with personally, and so far as I am aware only one patient showing signs of a similar though not identical nature has been so far recorded. The case referred to is that reported by Rihl[51]; the description of the case is meagre, but the venous curves show a condition of paroxysmal tachycardia and appear to substantiate the author's conclusion that during the paroxysms auricle and ventricle contracted simultaneously. The condition is therefore a very infrequent one.

The patient, a report of whose condition is now given, has been under continuous observation for many months, and, of the numerous observations which have been made upon him, attention will be chiefly directed to those which directly affect the general conclusions of the present communication.

* The term atrio-ventricular rhythm would perhaps be more consistent with our present knowledge; for while we may state with a fair degree of probability that such rhythms originate in the junctional tissues, their accurate location in the node is not possible at the present time.

CASE 15.—H. M., a labourer, aged 61, came to the out-patient department at the City of London Hospital on 4th October, 1909, complaining of pain in the left side of the chest, shortness of breath and palpitation.

Past illnesses.—As a child he contracted measles; twelve years ago he states that he had "blood-poisoning" as a result of drain work (with fever, headache and vomiting). He was laid up for nine weeks. Five years ago he had rheumatic fever, but has not suffered from a return of this affection.

Present illness.—His symptoms date from March, 1909, when he was ill for three weeks with pain in the upper part of the stomach, loss of appetite, and vomiting. The vomit was white and contained slime; occasional clots of blood were ejected and the motions were black. The pain has continued in a less aggravated form but the vomiting has ceased. Discomfort in the epigastrium is experienced, amounting at times to actual pain of a lancinating character. The appetite is poor and the bowels are confined. Shortness of breath has been present throughout, and is especially prominent upon moderate or severe exertion. He sufferes from a sense of exhaustion, chiefly experienced in the legs. There is often a beating in the chest, more particularly when in the upright posture and after exercise. It has been absent at night, and is in abeyance while he is resting, but even when recumbent he has felt it at times. While standing he often becomes giddy and shaky in the limbs. He is losing weight.

Condition (4th October, 1909).—A poorly nourished, anæmic subject, who carries his years indifferently. There is general arterial disease; the arteries of the arms are conspicuous throughout the major part of their course. Systolic blood-pressure ranges from 110 to 150 mm. Hg.. There are no signs of pleuro-pericardial adhesions or aneurism. The pulse is of the Corrigan type and capillary pulsation is present. The right limit of cardiac dulness is 1 inch and the left 4½ inches from the middle line. There is a systolic murmur at the apex beat, which is forcible, and the murmur is well conducted to the axilla and left scapula. The second sound is normal at the apex. At the aortic cartilage there is a faint but definite early diastolic murmur. There are no physical signs pointing to gross lesion of the stomach.

The chief feature of the case is an irregularity of the heart, which consists of occasional or frequent extrasystoles, and of short paroxysms of tachycardia. The interruptions of the otherwise regular rhythm are always present in some degree; they are usually frequent when the patient stands, but much less frequent while he is reclining. If examined in the standing posture the short paroxysms are always found, if laid on his back they promptly disappear, and for several hours the irregularities may be absent. But if at any time he stands again there is a return of the fleeting periods of tachycardia. Change of posture has been frequently ascertained to be almost invariable in its effects. The paroxysms do not occur in the sitting posture, and therefore cannot be ascribed to the action of gravity upon the heart. They are not present after or during exertion, or long periods of suspended respiration, in the recumbent posture. The postural observations have recently been extended by Dr. Marris of St. George's Hospital. He finds that there is a critical angle, during the change from horizontal to vertical lie, at which the ectopic beats appear. He has further observed that a tight abdominal binder abolishes the irregularity which prevails in the erect posture. Venous filling, or more properly the lack of it, therefore appears to be an important contributory factor in the production of the abnormal mechanism.

The reaction of the heart to posture has necessarily resulted in difficulty in obtaining venous curves. The paroxysmal venous curves have been won

on three occasions only. It has also rendered the simultaneous record of venous and electrocardiographic curves impossible; the electrocardiograms were taken with the patient standing. The paroxysms and single interruptions are immediately recognised by the patient when they occur.

The pulse, when regular between the paroxysms, is usually slow, its rate varies between 37* and 70 per minute. At times there is a certain degree of sinus arrhythmia. The extra or ectopic beats which interrupt the normal rhythm are of varied form, but most of them conform to the recognised pictures of auricular and "nodal" extrasystoles, when examined in the venous curves. The electrocardiographic tracings show at least four varieties, but as their nature is not entirely clear further reference to them will be postponed. More frequently than otherwise they differ from the beats of the paroxysm itself. In this respect they resemble the single interruptions met with in other cases of paroxysmal tachycardia.

The paroxysms consist of a series of regularly placed beats (there is some general diminution of rate as the individual attack proceeds), and the rate lies between 135 and 160 per minute. Alternation is very frequent, and in simultaneous electrocardiographic and radial curves the small beat in one may correspond to the large beat in the other, or vice versa. The onset of the tachycardia is marked by the occurrence of several anomalous beats which do not properly belong to it. In the radial curves (Figs. 27-28) they are differentiated by the low level which they occupy in the curve, and by their relative insignificance. Venous curves (Fig. 27, second paroxysm) may point to their having a higher origin† than the beats of the paroxysm proper. The high level of origin is also borne out by the electrocardiographic curves (Fig. 26), as will be seen later.

The contractions of the heart during the paroxysm proper yield venous curves which consist of high peaked waves, similar to those seen when auricle and ventricle are known to contract together (e.g., in complete heart-block and in cases of single ventricular extrasystole). The points at which *a* and *c* are estimated as due do not fall absolutely together, but they are nearer together than is the case with certain of the premonitory beats. The length of the interval is generally 0·06 sec., the *a-c* interval of the normal rhythm is 0·2 sec.. (The presphygmic interval for normal and paroxysmal beats is the same on the arterial side). The venous curves definitely indicate that auricle and ventricle are in simultaneous contraction. Yet the auricular contraction commences at a slightly earlier time than does that of the ventricle. The interval is too short to permit of the conclusion that ventricle is responding to auricle, but, on the contrary, it may be supposed that the two chambers are contracting in response to a common source of impulse formation. It may be argued, from the evidence, that this common focus lies nearer to the auricle than to the ventricle. In other words, the

* This low rate occurred during sinus arrhythmia. The usual rate is 50-54.

† The higher the origin the greater is the *a-c* interval.

facts suggest that the impulses are derived from the neighbourhood of, or actually arise in, the node of Tawara (the junction of auricle and bundle).

The relationship of the auricular and ventricular systoles is borne out by the electrocardiographic curves. Comparing the beats of paroxysm and normal rhythm in Fig. 26, the essential difference between them lies in the shape of the variation which directly precedes R (the first event of the ventricular cycle); in the normal curve this variation P is recognised from its presystolic situation to be the result of auricular systole. In the paroxysmal beat a variation of similar extent is observed, but it is directed downwards instead of upwards. The complete inversion of P during the paroxysm very strongly suggests that the wave of contraction in the auricular walls has travelled in a direction the reverse of the normal. In previous communications I have already referred to the importance of these abnormal P waves, and to their significance as indicating an alteration of direction of beat and therefore a dislocation of the site of impulse formation upon which they are dependent. In the present instance it may be concluded that instead of passing from above downwards the wave of contraction has travelled from below upwards*. The interval P-R in the normal beat is 0·14 sec. and in the abnormal or paroxysmal beat it is 0·08 sec.. The difference between the two is less than would be anticipated from the venous curves, but both the nature of the abnormal P variation and the closing up of the P-R interval confirm the previous conclusion drawn from polygraph curves, namely, that the contraction of both auricle and ventricle is derived from a single impulse generated between auricle and ventricle, but rather nearer to the former than to the latter.

A fortunate experimental observation has placed both the measurements and the interpretation upon a satisfactory footing. In the figures so far discussed we have no simultaneous venous and electric curves, and the absence of such curves, curves which would have proved of value, is the necessary outcome of the postural changes of rhythm found in the patient. But the curves actually shown were taken within an hour of each other, and one may be read into the other. The experimental observation, to which we may now refer in detail is of particular value, not only because the electrocardiographic curves are identical with those already shown, but because in this instance we have simultaneous curves from auricle and ventricle, taken directly from the heart wall by means of myocardiographic levers. Fig. 29 consists of ventricular, auricular and electrocardiographic curves. It is an example of fibrillation of the auricle, and its offset and return to the normal rhythm (of which one beat, the last, is shown). The first part of the curve has been discussed in an earlier section. Attention is now directed to the last four beats (R^{13} and R^{16}). The portions of the ventricular

* In speaking of up and down, I use the terms in the morphological, and not necessarily in the anatomic, sense; for we are as yet unaware of the direction of the anatomic pathway of contraction in the auricle.

electrocardiographic curves are alike in the four instances, and consist of R and T variations. It is in the presystolic event that the notable variation occurs. Preceding the ventricular contractions R^{13} and R^{14}, the waves P (P^{20} and P^{21}) stand in marked contrast to the normal auricular variation (P^{23}) of the normal beat (R^{16}). They are completely inverted*. In the case of beat 15 a transition form is seen and scarce a trace of P remains (P^{22}). The explanation is to be sought in the accompanying myocardiograms. The As-Vs intervals are marked by the vertical lines and stand at 0·43, ·043, ·066, and ·088 respectively. (The intervals have been measured and marked upon a greatly enlarged and photographic reproduction of the original curve.) (The commencement of the ventricular upstroke is not always as well defined as it might be, but the correctness of its position as marked may be readily checked by reference to the first notch of the plateau and to the peaks R.) The widening of the As-Vs interval occurs in two stages, and can be seen without measurement by glancing at the relative positions of the tops of the auricular myocardiographic curves and the bottoms of the ventricular myocardiographic curves. These simultaneous tracings demonstrate the mechanism of the heart beat corresponding to the electric curves in question in a conclusive manner. It is obvious that as an accompaniment of the decreased P-R interval (the figures stand at ·065, ·066, and ·088), auricle and ventricle are in synchronous contraction, during portions of their respective cycles. For each auricular systole is of the same length whether it belongs to the normal or abnormal cycle. The same type of auricular electric curve and the presence of shortening of the P-R interval in the clinical example permits us to conclude that a similar mechanism is present, and confirms the previous deductions.

The intervals in the experimental curve are subject to several further considerations. In the first place there is a notable discrepancy between the shortening of the As-Vs intervals and the corresponding P-R intervals. The intervals for beat 16 are alike (namely, 0·88 sec.). The shortening of the As-Vs interval of beat 14 is to ·043 sec., while that of the P-R interval is only to ·065. The discrepancy, or difference in shortening, is approximately 0·02 sec.. Attention to this discrepancy is of interest, and it meets with a ready explanation, which is in accord with the remaining remarks. It is due to the position of the myocardiographic lever attached to the appendix of the auricle. If, as would be anticipated, the auricular appendix enters contraction before that portion of the auricular musculature which borders on the ring, a difference in the intervals would be expected. In the normal and abnormal beats considered, let it be allowed that the impulse travels downwards in the one case and upwards in the other, then a transmission

* Similar electrocardiograms (experimental) have been published by Hering, (*Archiv f. d. ges. Physiol.*, 1909, cxxvii. 155) but in the absence of myocardiographic curves their interpretation could not be undertaken with any degree of certainty. Experimental extrasystoles started at the inferior vena cava yield inverted P variations, but the extent of the dip is not so great as that which is presented by these curves.

time of ·01 sec. for the single journey would suffice to explain the difference in the intervals. On one occasion I have obtained curves which bear out this explanation. Direct leads, with the electrodes of Gotch[13], were instituted from the base of the superior vena cava and from the upper end of the inferior vena cava, while the auricle was in fibrillation. Suddenly the fibrillation ceased spontaneously and was succeeded by several beats of the heart as a whole, in which the mechanism was abnormal, before the final normal rhythm and sequence were established. The normal beats of the re-established rhythm were accompanied by auricular contractions which yielded electric variations of constant excursion and direction, the main variation showing electro-negativity of the upper lead with a lesser swing in the positive direction following it. The abnormal beats on the other hand, in which the P-R interval was reduced, showed the reverse picture, the excursions remained the same but the direction altered from negative to positive and positive to negative respectively. In the case of the normal beat we have to assume primary* activity of the tissues abutting on the superior vena cava; in the case of the abnormal beat on the contrary we have equally to assume primary activity in the tissues in the neighbourhood of the inferior vena cava. All the evidence leads us to conclude therefore, that at times the contraction wave is temporarily reversed in the auricle when the latter breaks back from fibrillation to the normal response to the heart's pace-maker†.

Now a similar discrepancy has been noted in the clinical instance between the *a-c* and P-R intervals, but a like explanation will not apply. It is probably attributable to the abnormality of the contraction in the instance of the reversed beat, and to a later appearance of *a* in the veins of the neck under these conditions. The comparison is of chief importance in emphasising the greater accuracy of the P-R as opposed to the *a-c* interval in the clinical instance. The *a-c* interval during the paroxysm is not an absolutely true representation of the As-Vs interval, it is too short, and the electric measurement, ·08 sec., is the more accurate.

The parallel between clinical and experimental curves is striking, but if further evidence were required, it would be found in the relative heights of the peaks R in the two instances. The peaks R which follow inverted P variations are always higher, whether they are observed in the clinical or experimental example.

The experimental curves show us conclusively that we are dealing with simultaneous auricular and ventricular contraction. That there is only a small reduction of the normal As-Vs interval is immaterial from this point of view, but it is of importance in demonstrating that the level

* Referring to superior and inferior vena cava only.

† Winterberg[67] has made certain observations which tend to the same conclusion. The auricular impulses, at the escape from fibrillation, are not necessarily generated in the "sinus," they may arise in other portions of the auricular walls.

of impulse formation is relatively high so far as the ventricle is concerned. The main delay is in the bundle itself.*

The close analogy between the two sets of curves allows of similar conclusions in the clinical instance. The difference in the P-R intervals of normal and abnormal beats in the patient is greater than in the experiment. But the auricle of man is larger than that of the dog. The normal P-R interval in the patient is nearly twice that found in the animal.

Returning again to the clinical case, in Fig. 28 it will be seen that there is irregularity during the paroxysm itself, and that this irregularity is due to the presence of premature beats interrupting an otherwise regular rhythm. One of the interrupting beats has been caught electrocardiographically and is shown in Fig. 30. The extra beat is of the type recognised as due to an extrasystole of the left ventricle. Now the pause which follows the premature beat is fully compensatory, and this in itself is valuable confirmatory evidence of the auricular origin of the ectopic rhythm which it disturbs. The supraventricular origin of the paroxysmal beat is also shown by the shape of the ventricular electric complex.

Another point of interest in the clinical curves is well seen in the first escaped beat of Fig. 26. Here neither normal nor inverted P variation is present. Such heart cycles have been of common occurrence in this patient at the termination of paroxysms, and many such beats interrupt the normal rhythm. A complete parallel is found in beat 15 of Fig. 29. One may infer from an examination of the intervals, if not from *a priori* arguments based on the isoelectric interval directly preceding the peak R^{15}, that this is an example of a transition form between beats 14 and 16. That is to say, that we are dealing with a beat which starts at a higher level than beat 14, and at a lower level than beat 16. In brief, it has its origin at a point lying between the normal pace-maker† and the lowest or junctional level of the auricular tissue. The same conclusion applies to clinical beats of a similar nature seen in Fig. 26. Of the escaped beats in Fig. 26, the first and second probably belong to this category, while the third is of the same nature as the beats of the paroxysm.

* That auricle is not giving the pace to the ventricle is certain, for the following reasons:—An increased conductivity would not be expected during the clinical paroxysm, for the rate is increased. Neither would a gradually decreasing conductivity be anticipated in the escaped beats following the fibrillation, for the rate is lowered and blocking of some of the fibrillary impulses was previously present.

† For the purposes of the argument there is no need to place the normal pace-maker of the heart in the neighbourhood of the superior vena cava, but as a matter of fact the evidence as a whole points in this direction. In a single experiment, using direct leads from various parts of a dog's auricle, it was found that the neighbourhood of the superior vena cava becomes electronegative or active before either appendix, inferior vena cava or a point of musculature at the groove directly below the superior vena cava on the front of the heart. In several experiments it was found that the auricular electric complex of extrasystoles induced in different parts of the auricle shows notable variations. The auricular complex of extrasystoles originating near the superior vena cava resembles the normal complex most closely. This evidence points very definitely to the presence of the pace-maker in the neighbourhood of the superior cava.

The conclusion that in the clinical case paroxysms are present, and that each paroxysm consists of a rhythm created by impulses which are derived from the lower levels of the auricular tissue, or, in other words, in the neighbourhood of the node, depends upon the evidence which has been given and which may be briefly summarised as follows*:—1) Upon the shortened *a-c* and P-R intervals. The extent of the shortening and the circumstances under which it occurs (pulse rate, etc.) preclude the possibility that ventricle responds to auricle.

2) Upon the complete reversal of the electrical variation accompanying the auricular contraction.

3) Upon the presence of a normal type of ventricular electric complex, and the occurrence of the complete compensatory pause following the interruption of the ectopic rhythm by a ventricular extrasystole. These facts indicate the supraventricular nature of the rhythm.

4) Upon a close comparison with duplicate curves obtained experimentally, and upon the more accurate analysis which could be undertaken in the latter.

It is a matter of importance that it should be recognised that such a phenomenon as "nodal rhythm," in the broad sense of the word, does exist in clinical medicine; it is also imperative that its rarity should be acknowledged. And further, and most important, it must be understood that the picture presented by the only cases exhibiting it, of which we have knowledge, is entirely at variance with that of the condition which has hitherto passed under this term. "Nodal rhythm," as we now know it, is a rhythm in which there are definite signs of simultaneous auricular and ventricular contraction; *a fortiori* the rhythm is regular. It falls into line with the examples of fast and paroxysmal ectopic rhythms, arising at different levels; in these also the individual beats can be shown to spring from a single and fixed focus. It may be taken as a general rule that a paroxysmal rhythm, when regular, arises as a result of impulses generated in a single focus. The converse, that irregular paroxysms are generated from several foci, only applies if such paroxysms are fast, and if irregularities resulting from conductivity and contractility changes can be excluded.

In conclusion it is my pleasant duty to acknowledge my gratitude to those who have brought material for observation to my notice, and to those who have facilitated the examination of such material. I am indebted to all the members of the medical staff, honorary and resident, at University

* Before leaving this patient several points must be referred to which are in harmony with observations upon other cases of ectopic paroxysm, and they have been utilised in the discussion upon the subject in a paper published in the last number of this *Journal*. In the first place there is the presence of the premonitory contractions at the commencement of the paroxysms, and these are of a different nature to those obtaining in the paroxysm itself. Secondly, there is the fact that the beats which disturb the heart's regular action between the paroxysms are as a rule of a different form to those of the paroxysm. Thirdly, there is the marked depression of rate with subsequent acceleration at the cessation of the paroxysm (clearly seen in Fig. 28).

College Hospital. My thanks are especially due to Dr. James Mackenzie and to Dr. John Rose Bradford.

Chief conclusions.

1. Amongst the many forms of persistent irregularity of the human heart, none is more common than that which may be termed complete irregularity of the heart. It is accompanied by an absence of all sign of normal auricular action ; and it is due to fibrillation of the auricle.

2. Auricular fibrillation as a pathological and clinical entity exhibits certain definite signs. Amongst the distinguishing features the following are the most important.

a) Absolute irregularity of the ventricle.

b) The ventricular form of venous pulse.

c) A characteristic electrocardiogram, in which the ventricular curve is of the usual type, but in which the normal auricular representative is replaced by a series of rapid oscillations, which are superimposed upon the rest of the curve and deform it. The oscillations are generated in the auricle, and are the result of the fibrillation.

3. Digitalis retards the ventricular rate in clinical auricular fibrillation by enhancing a previously existing auriculo-ventricular heart-block. The rapid and irregular impulses showered upon the ventricle from the fibrillating auricle are hindered in their passage from one chamber to the other by the action of drugs of this class. The influence of digitalis is exerted, directly or through the vagus, upon the junctional tissues.

4. Auricular fibrillation in man may be accompanied by heart-block of all grades, and the heart-block may or may not result from digitalis administration. When the heart-block is complete the ventricular action is slow and regular.

5. A rhythm arising in the neighbourhood of the node of Tawara is a real clinical phenomenon. It is distinct from the mechanism which produces complete irregularity, and it is a rare affection.

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Fig. 12. ⅓ linear. Two photographic curves from a patient with mitral stenosis (*CASE 11*) taken before the onset of fibrillation. The electric curve, E, and the radial tracing, Ra, show irregularities interrupting the sinus rhythm. They are auricular extrasystoles. The P-R intervals are increased. In the second curve successive auricular extrasystoles are shown.

Fig. 13. ⅓ linear. From the same case, a few days after the onset of fibrillation. *I*, lead from right arm and left leg. *II*, from sternum at junction of second cartilage and from apex. *III*, from sternum and point over right auricle. *IV*, from the third left space in anterior axillary line and from apex. *V*, from apex and abdomen, 3 inches internal to the apex and just below the ribs. The first electrode site mentioned above is in each case the site of the arm electrode.

Fig. 14. ⅓ linear. From a patient with mitral stenosis of rheumatic origin. Completely irregular heart action and the ventricular form of venous pulse were present. The figure shows electrocardiograms obtained by six separate leads. I, right arm and left leg. II, sternum between junction of second and third cartilages, and point over right auricle. III, sternum and apex. IV, sternum and outer end of first intercostal space. V, outer end of third intercostal space and apex. VI, apex and point on the abdomen, 3 inches internal to the apex and just below rib margin. The first electrode site mentioned above is in each case the site of the arm electrode. The curves show that the oscillations are maximal according to the proximity of electrodes and superficial auricle. They also show the normal character of the ventricular complex.

Figs. 15 and 16. Electrocardiographic curves from a case of paroxysmal irregularity. Fig. 16 was taken two days after the subsidence of a paroxysm. Fig. 15 demonstrates the absence of regularity; the characteristic oscillations; the deformity of the variation T; the absent relationship between height of peaks R and corresponding radial beats. There is no sign of a normal P variation. Fig. 16 shows the return of P and the shortening of the peaks R. (*CASE 10*).

Fig. 17. Electrocardiographic and Hürthle carotid curve from a dog while the auricle is fibrillating. In the early part of the curve the heart is recovering from vagal stimulation. The curve shows the oscillations unmasked by slowing, and later the characteristic deformity of the curves joining adjacent R peaks, and the absence of relationship between the height of electric peaks and arterial curves.

Fig. 18. ⅓ linear. Electrocardiographic curves from a patient with complete heart-block and auricular fibrillation (*CASE 13*). *I*, right arm and left leg. *II*, both electrodes over large right auricle. *III*, sternum and right auricle. *IV*, sternum and apex. *V*, apex and abdomen, internal to nipple and just below rib margin. The electrode site first mentioned, is in each case the site of the arm electrode.

Fig. 19. × ⅓ linear. Venous and electrocardiographic curve from a patient with bradycardia. Oscillations of the same rate are present on both curves (*CASE 12*).

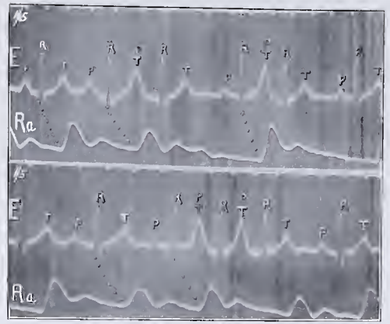

Fig. 12.

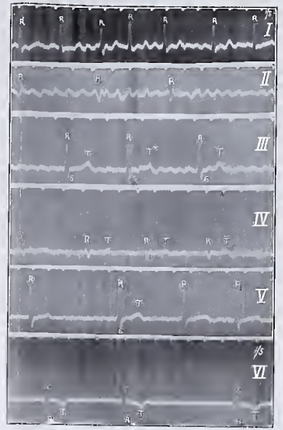

Fig. 14.

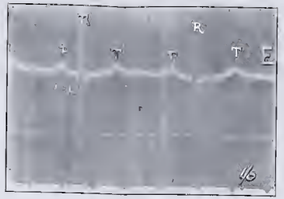

Fig. 16.

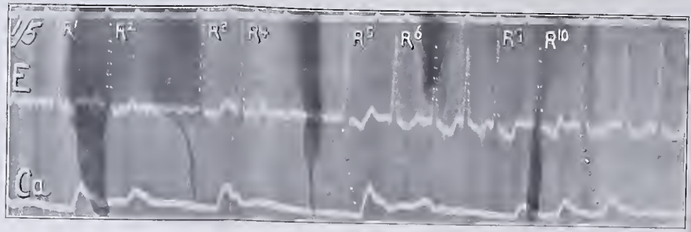

Fig. 17.

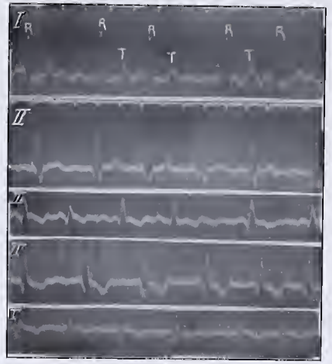

Fig. 13.

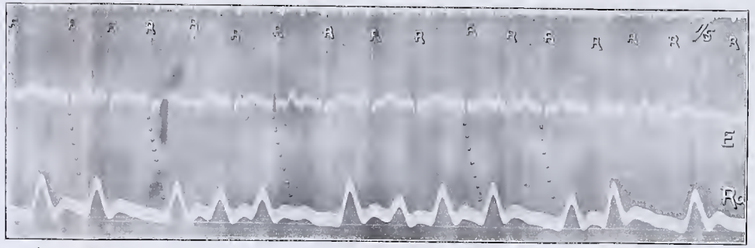

Fig. 15.

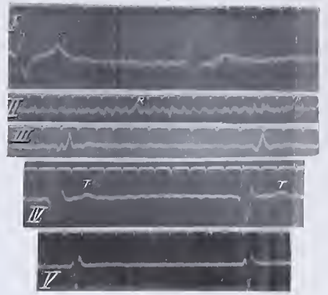

Fig. 18.

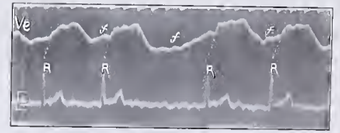

Fig. 19.

Fig. 20. Experimental curve. Taken from a dog with chest wall intact, while the auricle was fibrillating. Venous and electrocardiographic curves are shown. The venous curve is ventricular in form; the electrocardiogram shows irregularity of the ventricle, and well marked auricular oscillations.

Fig. 21. From the same animal a few minutes later. The venous pulse is now auricular in form; the electrocardiogram shows the return of P, while the oscillations have vanished with the fibrillation.

Fig. 22. Electrocardiogram from a dog with intact chest wall, showing the oscillations in a characteristic form. The tall peaks R are those which fall with the summits of oscillations. For comparison with Fig. 23.

Fig. 23. An electrocardiographic curve taken from a patient with mitral stenosis. The pulse was completely irregular and the venous pulse ventricular in form. For comparison with the experimental curve shown in Fig. 22.

Fig. 24. - $\frac{2}{3}$ linear. Electrocardiographic and radial curves from a patient with complete irregularity of the heart and a venous pulse of the ventricular form. The beats of the ventricle are in response to auricular impulses, and also to inherent impulses (ventricular extrasystoles). *CASE 7.*

Fig. 25. Electrocardiographic curve from a patient (*CASE 14*) with the ventricular form of venous pulse. The ventricle beat regularly. P is fully developed.

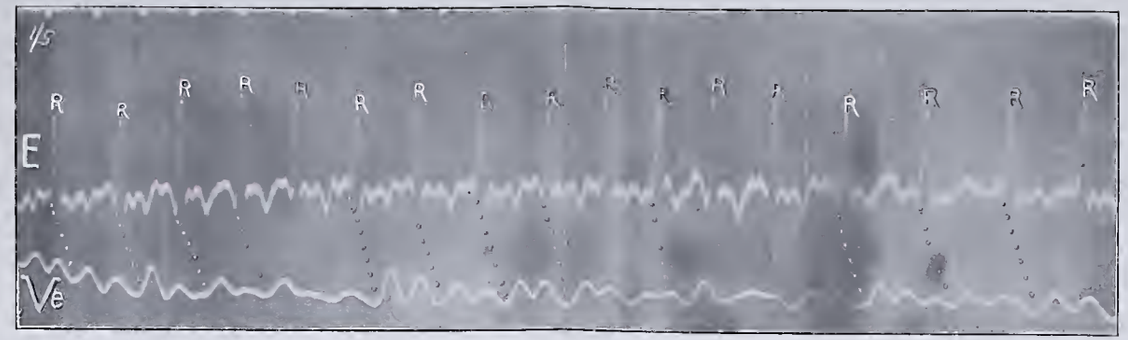

Fig. 20.

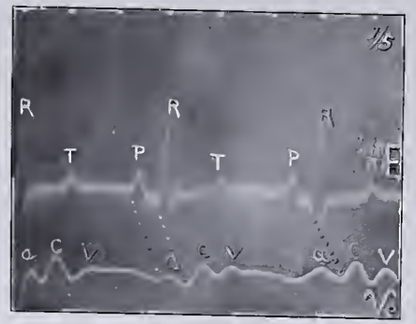

Fig. 21.

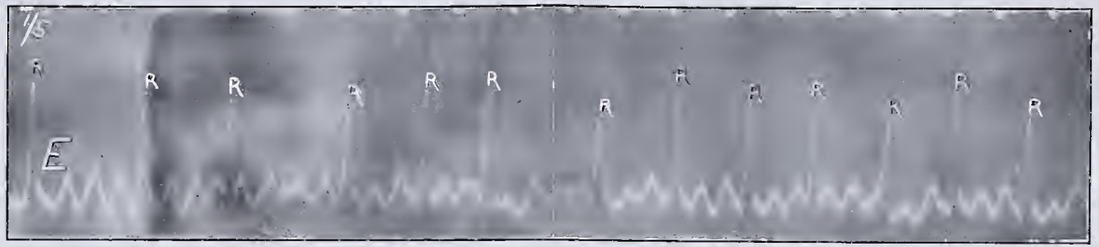

Fig. 22.

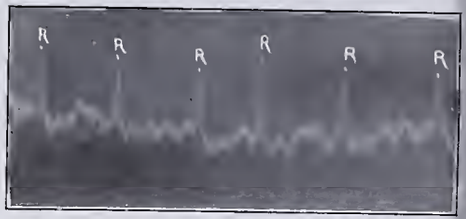

Fig. 23.

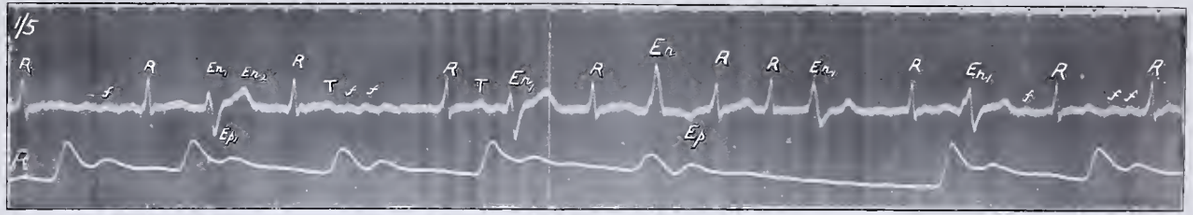

Fig. 24.

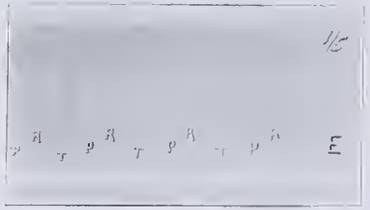

Fig. 25.

Fig. 26. Electrocardiographic and radial curves from a patient with paroxysmal tachycardia (*CASE 15*); a complete paroxysm is shown. The curve opens with three normal beats accompanied by the usual P, R and T variations. These are succeeded by two auricular extrasystoles and then the true paroxysm commences. The beats of the paroxysm are like those of the normal rhythm, except that R is higher and T lower, and the variation P is inverted. The paroxysm ends in a long pause and this is followed by two abnormal beats, a paroxysmal beat and two normal beats in which the upright P variation has returned.

Fig. 27. Venous and radial curves from the same case (*CASE 15*), showing two complete paroxysms. The a-c interval is reduced to 0·06 sec. during the ectopic rhythm.

Fig. 28. Apex and radial curves from the same case (*CASE 15*), showing three complete paroxysms. The beats preceding the paroxysm are well shown in the first two attacks. The presphygmic interval is the same during slow and fast rhythms. Two premature beats are seen in the third paroxysm, the pauses are completely compensatory.

Fig. 29. Simultaneous curves, myocardiograms from ventricle and auricle and electrocardiographic, from a dog. The ventricular beats in the top curve are marked 1-16, and the corresponding R peaks are similarly marked. The auricular vibrations and contractions are marked (P^{1-28}). The incidence lines were drawn and the measurement made on a greatly enlarged photographic reproduction. The subsequent reduction, to 6/7·5 the size of the original curve, could not be accomplished without retouching the background.

The early parts of the curve show auricular fibrillation and allow a complete analysis of the electric curve into its component auricular and ventricular elements.

The second part of the curve, following the return of the auricle to co-ordinate contraction, is given for comparison with Fig. 26. It shows the exact time relationships of contraction of auricle and ventricle when anomalous P variations are produced.

Fig. 30. × ⅓ linear. (*CASE 15*). The patient was trembling when the curve was taken. One of the premature beats mentioned in the explanation of Fig. 28, and interrupting a paroxysm, is shown. The pause is compensatory and the beat itself is recognised as an extrasystole arising in the left ventricle.

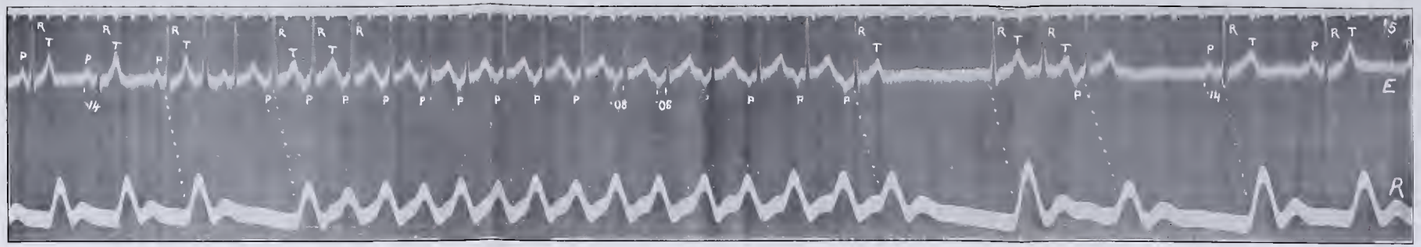

Fig. 26.

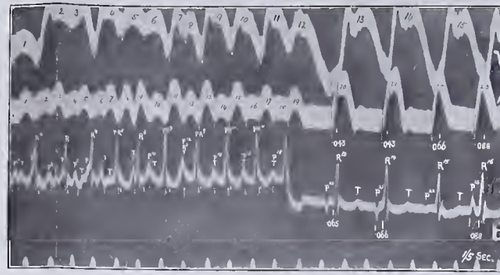

Fig. 29.

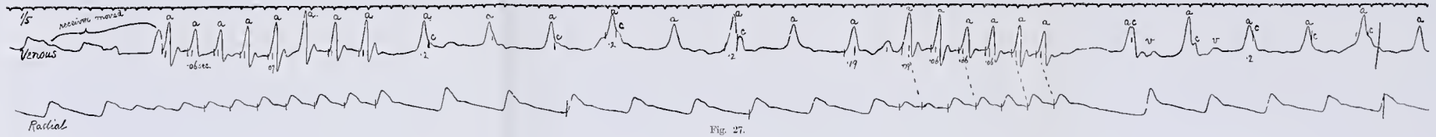

Fig. 27.

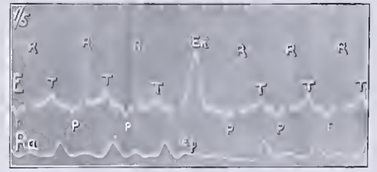

Fig. 30.

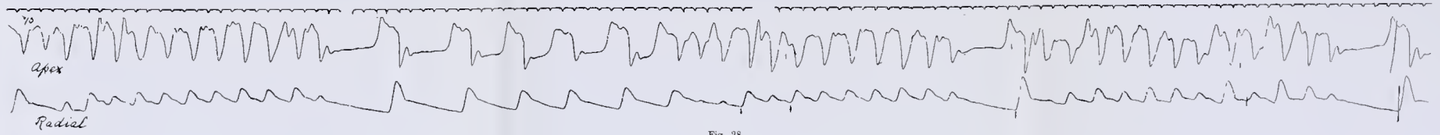

Fig. 28.

Fig. 31. A series of experimental curves from a single animal.

Curve *I*.

Leads from a point on the auricle just below the superior vena cava and from the inferior vena cava. The auricle was fibrillating up to the place marked by an arrow. At this point the co-ordinate contractions returned. A = auricular variations. V = ventricular variations.

Curve *II*.

A similar curve. The point where auricular fibrillation ceases is uncertain. In both curves the oscillations are maximal.

Curve *III*.

Leads from two points on the right ventricle, one above and near the right border, and one below and near the apex. The auricle is fibrillating over the whole curve, yet no oscillations are seen. Up to the point where the arrow is placed the vagus was stimulated and a slight escape of current is shown in the curve.

Curve *IV*.

From the same points after the return to the normal rhythm. A comparison with the last curve shows that the direction of contraction in the ventricle is identical while auricular fibrillation and auricular co-ordination are present.

Curves *III* and *IV* may be compared in respect of the amplitude of the opening variations of the separate beats. In *III* it is variable, in *IV* it is constant. Therefore the auricular oscillations are not entirely responsible for the variation in the height of the peaks R during fibrillation, for in *III* no oscillations are present. The height of the opening variations is greater in *III* than in *IV*.

Curve *V*.

Leads from the upper and lower ends of the wound. The wound travelled through the centre and whole length of the sternum. The upper electrode was upon the base of the neck, the lower one upon the diaphragm. The curve is very small and though much somatic musculature was included between the electrodes, no oscillations are seen. As a consequence, the oscillations are shown to arise in the heart itself. At the arrow the normal rhythm is resumed. Up to this point the sensitivity of the galvanometer was maintained at a constant point.

Curve *VI*.

The same leads as the last, but the sensitivity of the galvanometer is now increased approximately threefold. The oscillations are just visible and distort the curve to some extent. The normal rhythm is resumed where the arrow is placed. A careful comparison of the ventricular curves before and after the resumption of the normal rhythm shows them to be of essentially the same form, and this form is the normal one.

The electrode site first mentioned is invariably the site of the base electrode.

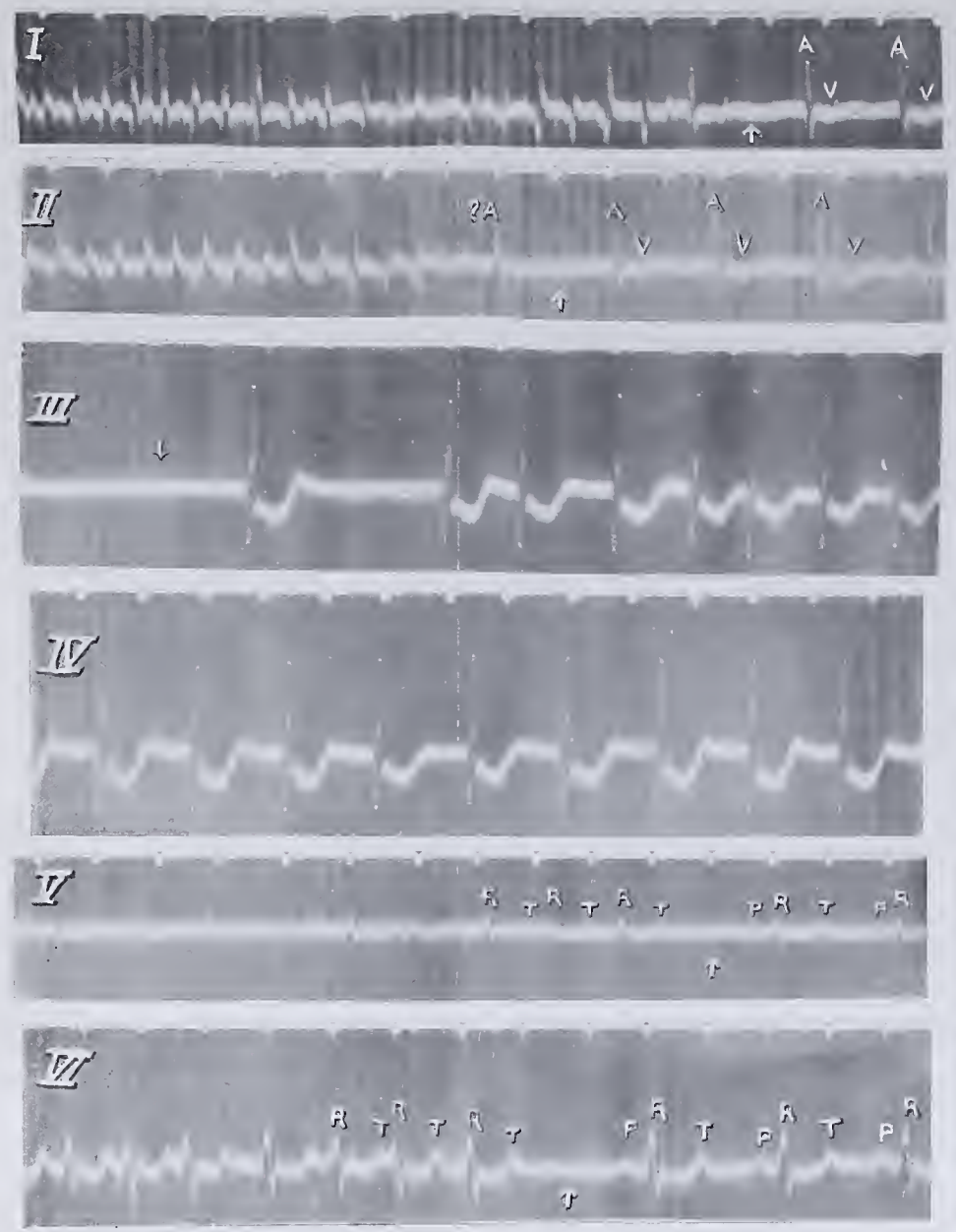

Fig. 31.